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San Francisco, California, June 17 - 20, 1954

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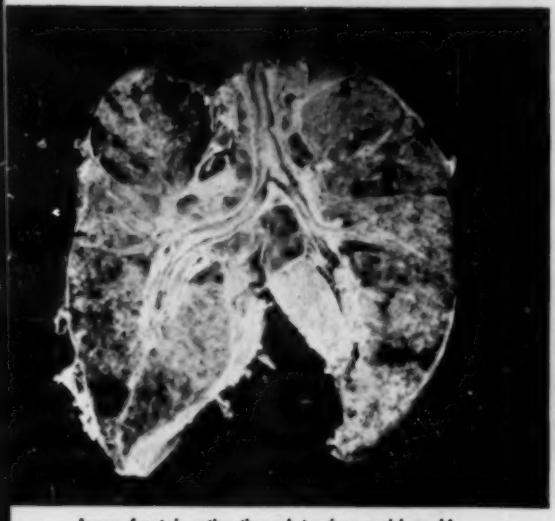

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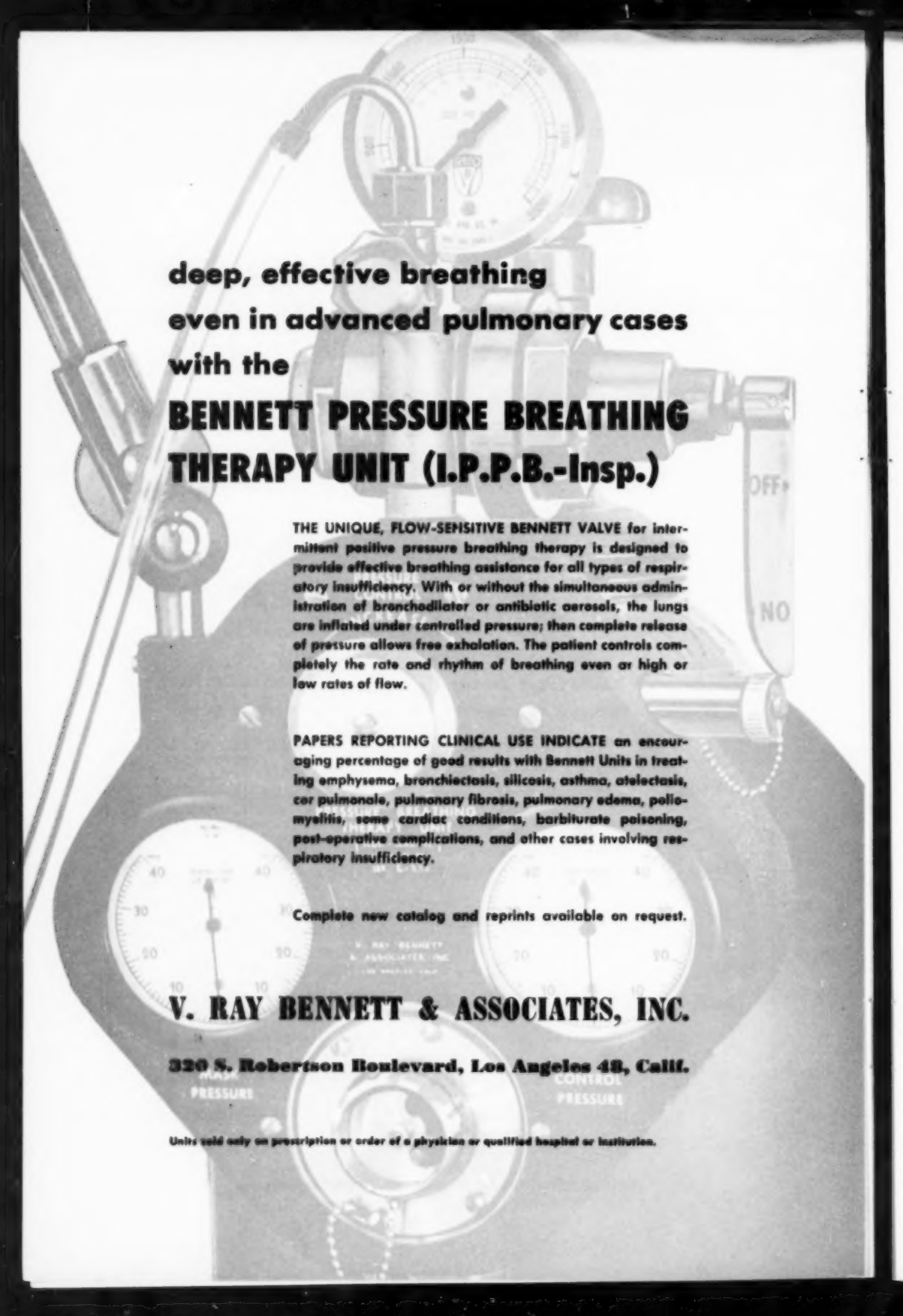

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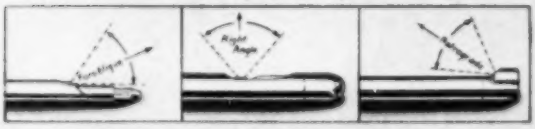

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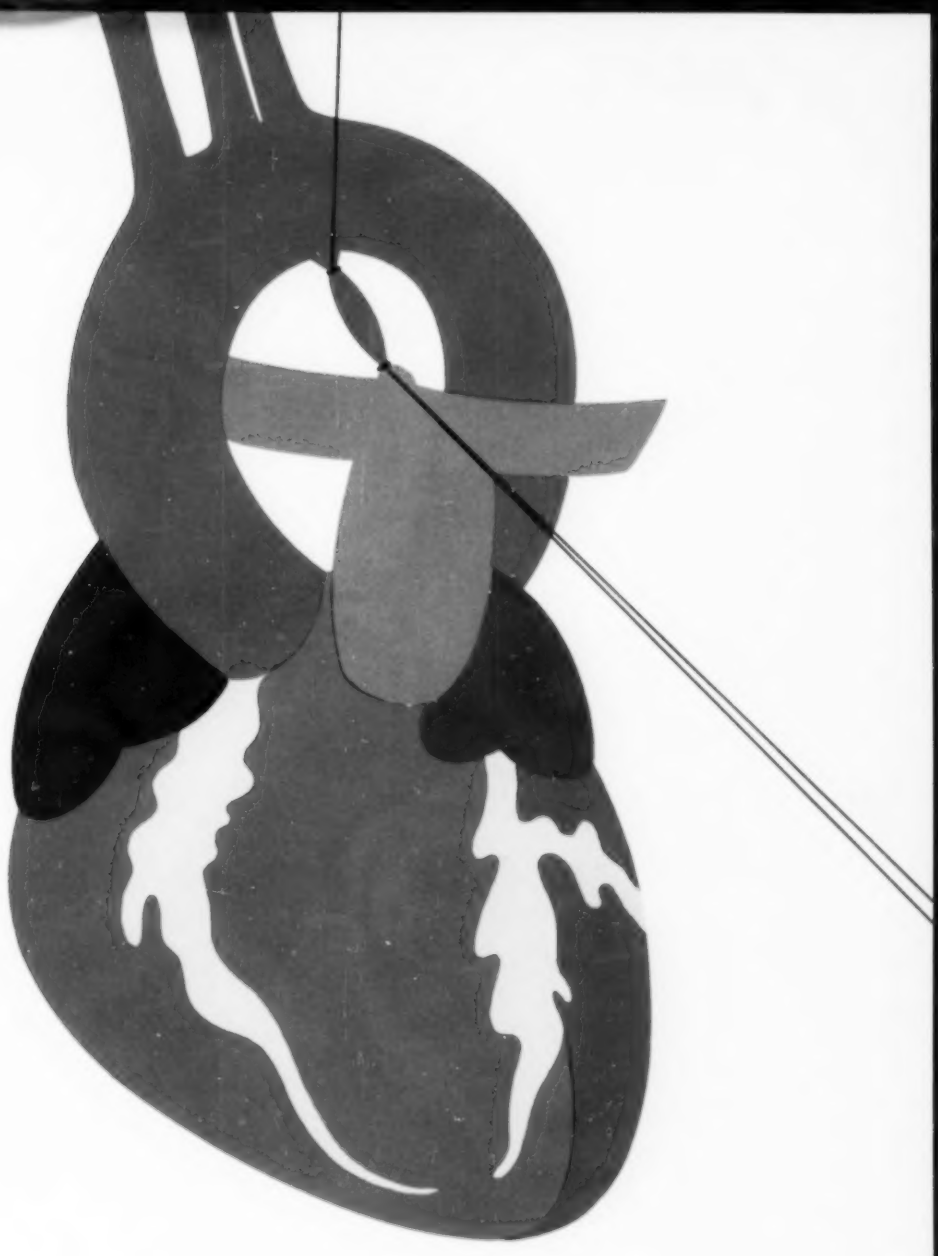

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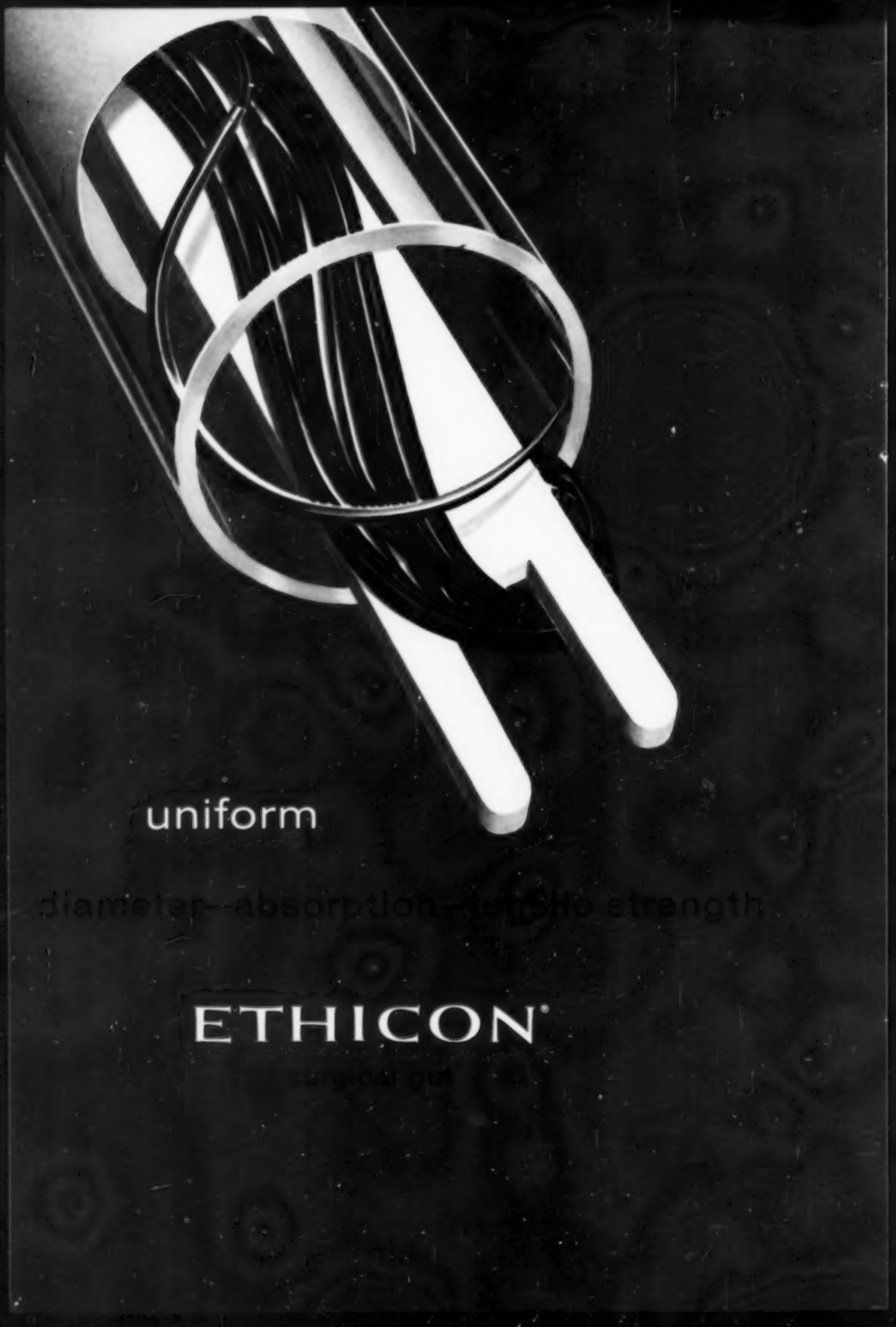
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The Problem of the Visceral Function of the Lungs

ISRAEL RAPPAPORT, M.D., F.C.C.P.
New York, New York

During the past two decades great strides have been made in the development of modern pulmonary physiology. But in these, some fundamental deficiencies have also been revealed indicating that current concepts are inadequate in explaining the increasing complexity of the recently observed phenomena of lung function and dysfunction. The point has now been reached when the need of a new concept, namely that of a special (visceral) function of the lungs is becoming increasingly apparent. Such a concept was proposed by the writer nearly two decades ago.[1]

Recent developments justify the assumption of a visceral function of the lungs, at least as a working theory, affording logical answers to some unsolved problems of pulmonary function and dysfunction.

In this discussion, four aspects will be considered:

I. The nonbiological character of present concepts of lung function.
II. Possible manifestations of visceral lung function as interpreted from the relationship between pulmonary structure and function.
III. Unsolved problems not adequately explained by prevailing concepts, but which logically point toward visceral lung function.
IV. Inferences regarding a specific hormone controlling lung function.

I. *The Nonbiological Character of Present Concepts of Lung Function:*

Analysis will show that prevailing ideas about lung function do not adequately explain recent observations chiefly because they are much too mechanistic. They labor under the presumption that all of lung function can be seen and measured in ventilation, circulation and gas exchange. The denial of a true organic function of the lungs implied in this presumption is its inherent defect. This peculiarly nonbiological character of modern concepts about lung function could hardly be better expressed than by the words of the late eminent physiologist Y. Henderson: "The lungs are peculiar organs in that they have little independent activity or self-regulation."[2] This obviously referred to the fact that since ventilation is the activity of the neuromuscular apparatus in the chest wall and diaphragms, since the pulmonary circulation is driven by the heart, and since pulmonary gas exchange is a physiochemical process taking place automatically, the structures of the lungs are not presumed to have any activity of their own.

Exception to this should be noted in current writings where the activity of one structure of the lungs is again being stressed, namely the role of its myoelastic apparatus in the elastic recoil during expiration. Many workers now look upon this as the sole and chief function of the lungs, which was well described in the following statement.[3]

"Mechanically the function of the lungs is more simple than that of any other vital organ . . . About half a million times during life in the act of inspiration the lung is passively stretched—half a million times in the act of expiration it must recoil—. This recoil is analogous to cardiac systole and is accomplished by virtue of inherent pulmonary elasticity . . . Failure of systole is the essence of the commonest form of chronic lung failure."

Reasoning from this the same author concluded logically by comparing the function of the lungs to that of "tire tubes of trucks." Far fetched as that may sound, it is yet true that current concepts have led to such an absurd conclusion of a mechanistic nature of lung function that comparison with lifeless rubber appears quite natural.

It should be noted here how the pendulum in the evolution of pulmonary physiology has swung from one extreme to the other. Biological thinking naturally led the pioneers in this field to the conviction that the organic function of the lungs should manifest itself in an active role in gas exchange. Hence Pflueger, Bohr, and Haldane were led to assume an active gas secretion by the alveolar epithelium. This idea was abandoned when Krogh, Bancroft and their followers demonstrated the purely physicochemical nature of gas exchange in the lungs. After that the pendulum swung from vitalistic far over to mechanistic thinking. In that transition from one to the other extreme a strange vacuum of ideas arose and remained quite unnoticed, as no idea came forward regarding some other possible role of the lungs' structures in gas exchange. Since then modern pulmonary physiology has become so deeply steeped in its nonbiological thinking that there is not even any awareness of the implication that the most vital organ of the body, next to the heart, is presumed to have no function of its own in the strict sense of that term.

II. *Possible Manifestations of Visceral Function of the Lungs:*

Because of lack of awareness of the meaning of visceral lung function it becomes necessary to present here briefly the writer's concept of this as proposed nearly two decades ago. This was intended to fill the vaccum in biological thinking about the function of the lungs as a vital organ of the body. It adheres to the principle that the organ-function of the lungs should have an active role in gas exchange. It proceeds from the fact that gas exchange in the lungs is ultimately a matter of the quantity as well as of the quality of the contact surface between blood and air in the air-spaces of the lungs. These considerations led to the idea that "creation of breathing surface" by the structures of the lungs is the essence of their visceral function. In detail this concept assumes that breathing-surface-creating activity is invested in the ultimate structural units of the lungs. This activity involves changes in the consistency of the semi-fluid membrane

covering the breathing surface, which allows the momentarily perfused capillaries to extend forward into the air-spaces. This activity of the breathing surface structures (capillaries and alveolar walls) results in simultaneous increase not only in the extent of the diffusing surface but also implies changes in its quality, i.e., permeability. These changes constantly serve to adapt the breathing surface to the momentary requirements of the body.

Some of the phenomena which may be logically interpreted as manifestations of visceral lung function of a type just described are as follows:

(1) *Intranatal establishment of lung function.* In the whole wide field of biology there is no more fascinating phenomenon, and no greater miracle wrought by nature, than the establishment of lung function during birth. The process by which this is brought about involves the following two cardinal changes: (1) The pulmonary capillaries upon first being perfused spring forward and with the force of the surging blood-flow, bulge into the air-spaces just created under the effect of the expanding chest. (2) The membrane covering the air-spaces so created undergoes changes in consistency and continuity allowing the forward extension of the capillaries. This process results in the creation of a breathing surface of such extent and quality as to permit the most intimate contact between the blood in the capillaries and the gases in the air-spaces. Thus the fundamental process implied in intranatal establishment of lung function is "breathing surface creation."

(2) *Relationship between structure and function* as applied to the lungs. Since it is the function of the lungs to afford the most adequate surface for efficient gas-exchange, conceivably the most purposeful activity of its structures must be one of actually creating such surface. It is an accepted biological principle that structure and function are always and everywhere intimately related. If surface creation is the true function of the lungs' structures, then this should be clearly indicated in the features of these structures. This principle is indeed valid for the structures of the lungs, perhaps more than in the case of most organs of the body. Structure and function are here but two facets of the same phenomenon whereby function begets structure and structure begets function. Function consists here of breathing-surface creation by structural changes which result in the creation of that surface. The structural manifestations of this are: (a) active bulging forward of the capillaries into the air-spaces. (b) Consistency changes in the breathing surface membrane. The former was emphasized

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Footnote: The concept of visceral lung function outlined above was published (Annals of Internal Medicine, March 1938) in a joint publication with Dr. J. A. Miller, who will be remembered as the founder of the famous research center for pulmonary function studies at Bellevue Hospital. Since this center has played a historical role in the progress of modern pulmonary physiology, it may be of interest to note here that Dr. Miller established this research center upon the initiative of the writer and that these concepts of "intrinsic lung function" inspired his deep interest in those lung function studies which have born rich fruit in the remarkable pioneer achievements of Dr. Cournand and his associates in cardiac catheterization, and in the outstanding work of Dr. Richards and his associates in pulmonary physiology.

by the observations of Macklin.[4] The latter has been stressed especially by Bremer[5] and confirmed in recent observations of Hayek[6]

These are naturally only inferences from features in fixed histological specimens. But, what we see of the lungs' ultimate structures in fixed histologic features should lead us to infer that they are mere reflections of a constant and extraordinary mobility implied in the type of structural activity assumed for "visceral lung function." It should be emphasized in this connection that this structural activity is more inaccessible to demonstration "in vivi" in the case of the lungs than in that of any other organ of the body. The lungs are the most dynamic organ of the body. Two significant facts should illustrate this point. (1) In spite of prodigious efforts we have never succeeded in visualizing the capillary circulation in the lungs "in vivo" by the methods which have been eminently successful in visualizing the capillary nets of all other viscera. This negative evidence has led Barclay[7] to ask the question "whether the capillaries of the lungs lying virtually among air-spaces at atmospheric pressure are different in some way from capillaries that are supported in solid organs."

The nature and the functional changes of the membrane covering the breathing surface has been under dispute among histologists for a long time. It may be present in one place and absent in the next, it may be a thick membrane in one place and a barely discernible thin diaphanous semi-fluid membrane in the next. The alveolar capillaries and the alveolar membrane have proved to be the most elusive structures in the body, facts which should lead us to infer that their activity is a most unusual one.

III. *Unsolved Problems of Pulmonary Physiology:*

Let it be emphasized at this point that the concept of visceral lung function is not intended to replace any of the established principles of pulmonary physiology. Visceral lung function is conceived as supplementing the established facts of ventilation, circulation and gas-exchange where these have proved inadequate to explain observations on pulmonary function and dysfunction. Admittedly the concept of visceral lung function is wholly theoretical and based on assumptions but these are applied to the part of lung function which has remained inaccessible to observation and measurement even by the most refined techniques of modern pulmonary physiology. Characteristically current explanations become inadequate when dealing with changes which take place in the structures of the lungs and indicate an active role of the latter in the local regulation of ventilation, circulation and gas-exchange. These unsolved problems of pulmonary physiology to say the least justify the concept of visceral lung function with all its assumptions as a working theory. Indeed, these unsolved problems point up the presumption implied in prevailing concepts that the lungs have no organ-function of their own. Perhaps the best examples of this are the following two basic problems of modern pulmonary physiology:

(1) Correlation of ventilation and circulation throughout all parts of the lungs.

(2) Correlation of diffusing capacity with momentary requirements of the body. Concerning these the following comments are to be made here:

(1) *Correlation of ventilation and circulation* throughout the lungs. Recent experimental and clinical studies have demonstrated precise correlation of ventilation and circulation throughout all parts of the lungs.[8-10] Implied in this correlation is the existence of a functional mechanism which operates in such a manner that blood-flow through various parts of the lungs is always distributed according to the efficiency of aeration; and vice versa the ventilation of the different parts of the lungs is always in proportion to the blood flow reaching the particular lung areas. The exact nature of this most remarkable functional mechanism remains to be determined. So much is obvious however that such precise local correlation of circulation and ventilation throughout all parts of the lungs must be based on a most purposeful function operating with the structures of the ultimate organ units. Involved in this function there must be a precise control of the lumina of arterioles and bronchioles under the effect of local tension levels of the gases in the blood reaching the capillaries and in the air inhaled into the air-spaces.

That such a correlation exists between ventilation and circulation throughout all parts of the lungs was predicted by the writer as far back as 1929 in the following statement: "It is a basic principle of pulmonary function that in any unit of the lungs there can be no circulation without ventilation and that there can be no ventilation without circulation It is this precise correlation between ventilation and circulation that underlies the mechanism set in action with all variations in respiratory requirements One must assume that this correlation is as precise in each unit of the lungs as it is in the whole of it."[11]

In contrast in prevailing concepts of modern pulmonary physiology it was taken for granted that nearly equal distribution of ventilation and circulation throughout all parts of the lungs is prerequisite for normal efficiency of gas-exchange. Maldistribution of ventilation and circulation throughout various parts of the lungs was therefore assumed to be the chief manifestation of abnormal function. Estimation of "distribution disturbance" became the clue to the study of pulmonary dysfunction.

It has been demonstrated that unequal distribution of the ventilation and circulation throughout various parts of the lungs is indeed the normal condition prevailing in the lungs. The significance of this has been described as follows:[12]

"Uneven ventilation is not necessarily a deviation from physiologically ideal conditions. Variations in the ventilation rate of different regions of the lungs when properly integrated with perfusion may well permit a respiratory adaptability to various conditions which would otherwise interfere with gas-exchange. The existence of uneven alveolar ventilation emphasizes that the lungs and breathing apparatus are not a simple or necessarily homogeneously functioning system. This indicates a potential inadequacy of many concepts which so assume, or which have arisen from methods that measure the net effect of the components. Physiological common-

places such as 'the alveolar' ventilation (or blood flow) is X liters/minute" and 'anoxia (or nerve action) contracts pulmonary vessels' represent net effects which may or may not be representative of events in all regions of the lungs."

Not only have prevailing concepts of lung function failed to explain the mechanism of correlation of ventilation and circulation unevenly distributed throughout the lungs, but as indicated by the above statement from a foremost worker these recently emerged observations cast doubt upon some of the basic tenets of modern pulmonary physiology. In passing it should be mentioned here that the existence of this mechanism has been questioned in turn by another leading worker[13] who recently asked "if such a purposeful, homeostatic mechanism regulating locally the alveolar ventilation—perfusion relationship exists—why in chronic pulmonary disease where this mechanism could be of great value, the lack of coordination between local ventilation and local perfusion is one of the main causes of physiologic disorders." The answer to this question is obvious from biological reasoning that it lies in the very nature of disease to interfere with normal regulation. Pathologic conditions will naturally result in disturbance of the normal correlation between ventilation and circulation in various parts of the lungs.

Such precise correlation of ventilation and circulation throughout all parts of the lungs operating with the ultimate structures of its organ units could hardly be conceived in any other way than by the assumption of a type of structure activity which practically implies "visceral lung function." It hardly needs to be pointed out, on the other hand, that it lies in the very nature of any concept of "visceral lung function" that ventilation and circulation throughout all parts of the lungs must be precisely correlated with it to secure its efficient operation.

Obviously such a functional mechanism implies regulation of structural (vasomotor and bronchomotor) activities in precise correlation with "visceral function" throughout all parts of the lung. Indeed the so-called N-lung experiments of Jacobaeus, about 15 years ago,[14] should have led us to suspect that the normal gas content of the air inhaled probably plays a role in this regulation. More recent observations[15] have actually confirmed the fact that the O_2 content of the air inspired and the venosity of the bolod returned to the lungs are factors involved in the correlation of ventilation and circulation throughout all parts of the lungs. All of which constitutes a true visceral autonomy in the manner of other visceral functions in the body. This visceral autonomy operates with such precision that venous blood returning to the lungs finds its way to the just freshly aerated breathing surface; and the fresh air just inhaled into the lungs finds its way to the capillary surface in which venous blood freshly arrived is most in need of gas exchange. It goes without saying that under pathologic conditions this functional mechanism becomes upset resulting in the manifestations well known to us from modern physiologic studies.

(2) *Correlation of diffusing capacity of the lungs with momentary gas exchange requirements of the body.* There is perhaps no other phase of

modern pulmonary physiology where the inadequacy of current views is more apparent than in the question of how diffusing capacity of the lungs is constantly adapted to the gas exchange requirements of the body. A review of the present status of this question in current literature should serve to illustrate the great difficulties into which mechanistic thinking has led in the solution of this problem. The following quotation from a most recent publication, perhaps best illustrates the point in question.[16]

"The most convincing evidence for functional changes in the pulmonary membrane is provided by the increase in diffusing capacity which occurs with exercise. The fact that such an increase occurs has been demonstrated repeatedly both in normal subjects and in patients with mitral disease. The explanation would seem to require either that the permeability to oxygen of the diffusing surface increase with exercise, or that its area broaden. While no proof is available, it seems likely that the area of the perfused portion of the pulmonary capillary bed might increase, but unlikely that the diffusion characteristics of the capillary walls and alveolar membrane should change. From these reasonable assumptions it is postulated: 1) That during exercise the area of the active diffusing surface increases; 2) conversely, that part of the surface, across which diffusion takes place during exercise, is inactive in the resting state; and 3) that this reduction results, in large measure, from the cutting off of blood flow through portions of the capillary bed.

"A way in which such dynamic changes in the diffusing surface might be brought about is suggested by the work of Burton, Nichols, Girling, Jerrard, and Claxton. These workers inferred, from careful studies of the circulation of the rabbit's ear and hind limb, that small vessels close completely at pressures below a certain level, called the critical closing pressure. They also found evidence that the critical closing pressure rises as the tone in the vessel walls increases.

"If these ideas are applied in the case of mitral stenosis, it would seem possible that some of the small pulmonary vessels close in the resting state because the pressure tending to hold them open is less than the critical closing pressure. As a consequence, blood flow through some of the capillaries may be eliminated with resultant reduction in the area of the active diffusing surface and reduction in the diffusing capacity as determined. Such a mechanism would account for the dynamic changes in the capillary bed which were postulated to explain the difference between the diffusing capacity at rest and during exercise. *Footnote:* These mechanisms would apply to normal as well as pathological states.

"The objection may be raised that the pulmonary vascular pressures are high in mitral stenosis and that, therefore, all vessels must necessarily be open even in the resting state. To this it could be answered that the pulmonary vascular resistance is also high in mitral stenosis, suggesting that the tone of the small vessels, and hence their critical closing pressure, is high. Probably the balance between the pressures tending to hold the vessels open and the vasomotor tone tending to close them determines which vessels remain patent and which do not.

"It may be postulated that gas exchange takes place across virtually all capillary walls during strenuous exercise, and that the diffusing capacity, determined under these conditions, is related to the structural characteristics of the entire diffusing surface of the lung. If this is so, the highest value obtainable during exercise, called the maximal diffusing capacity, may be interpreted in static morphologic terms. Values obtained at rest, on the other hand, are almost certainly affected by dynamic, as well as static, factors. It is therefore felt, that quantitative interpretations regarding the over-all structure of the diffusing surface are not justified by knowledge of the resting diffusing capacity alone."

This latest expression of current views on the functional activity of the diffusing surface was quoted in full in order to emphasize the following significant points: 1) Current views involve as much speculation about the changes in question as does the concept of visceral lung function.

2) These views are based on a new concept of a capillary mechanism the aplication of which to pulmonary capillaries is more than questionable, for there is reason to believe that these capillaries are perhaps unique in the body.[7]

3) These observations indicate that the changes in diffusing surface by which diffusing capacity is increased normally occur even under such (pathologic) conditions in which it is most difficult to visualize increased capillary perfusion. It would seem that under the circumstances in question these observations are better explained by qualitative changes in the diffusion surface than by increase in capillary perfusion.

4) There are still other gas exchange phenomena which are difficult to explain on the view that functional changes of the diffusing surface can be only quantitative in nature. Perhaps the most important of these which has as yet received little consideration is the nature of the difference in individual diffusing capacity, i.e., the individually different diffusing constants. Why do some individuals accomplish the same amount of gas exchange with less circulatory and ventilatory effort than others even while at rest and particularly during exercise. To say the least these differences in diffusing capacity are not adequately explained by merely quantitative changes, they strongly point toward changes in the quality of the diffusing surface.

The above quotation of current views on the functional changes in the diffusing surface illustrate furthermore the prevailing trend to dismiss without any explanation, as unlikely, the possibility of qualitative changes in the diffusing surface. It is not even considered necessary to explain why it is unlikely that the functional changes in the alveolar membrane could involve variations in the diffusion characteristics such as to permit changes in the permeability to oxygen of the diffusing surface in accordance with the momentary needs of the body.

It should be noted that the occurrence of changes in the alveolar membrane which alter its permeability to oxygen have long been proved and accepted as the most important pathologic change in pulmonary diseases in which its diffusing capacity is decreased. It is a long recognized prin-

ciple that pathologic changes differ from the normal only in extent but not in kind. Why then is it held inconceivable that changes of the alveolar membrane in the opposite direction which would increase its permeability to oxygen could ever occur as a part of normal lung function? The reason for this is not apparent unless it is the strong trend to express all biological phenomena in strictly mechanistic terms. As Einstein has recently described this trend, "Physics has often seduced the biologist into interpreting biological phenomena too primitively."

If we accept the principle that functional changes in the structures of the breathing surface serve to adapt it to the momentary requirements for gas exchange, it becomes logical to assume simultaneous changes in quality as well as in quantity. This is precisely what is assumed in the concept of visceral lung function. Considerations already briefly mentioned before led the writer to stress the assumption that not only is the number of perfused capillaries, i.e., their area of exposure increased, but also that as these capillaries spring forward into the momentarily active air spaces the semifluid membranes of these is being thinned out so that the interface separating blood and air permits the most intimate contact between them. Thus the diffusing surface and its permeability to oxygen increase simultaneously.

There is reason to believe that variations in the proportion of qualitative to quantitative changes will probably account for individual differences in "diffusion constants." By the same token it will explain the great individual differences in the ability to increase diffusing capacity during exercise.

Present knowledge of the gas exchange process in the lungs we believe, justify the following two assumptions: (1) There exists a special functional activity of the breathing surface structures which serve to adapt the diffusing surface constantly to the momentary gas exchange requirements of the body. (2) This activity is of a type involving changes in quality (permeability) as well as in quantity (extent) of the diffusing surface.

IV. *Indications for a Hormone of Lung Function:*

The manner in which lung function is regulated according to the needs of the body and correlated with cardiocirculatory function under normal and under abnormal conditions gives ample indication of what is often referred to as the "wisdom of the body." This regulation and correlation of visceral functions is known to be mediated by the action of hormones specific for each of the participating organs. Visceral lung function is to be conceived as analogous to other visceral functions. Like renal and hepatic functions, lung function implies in addition to the visible and measurable phenomena (ventilation, circulation, gas exchange) some as yet unrevealed subtle functional mechanism the seat of which is in the structures of its ultimate organic units (capillaries and membranes of alveoli), the activity of which is under autonomic control mediated by a specific hormone acting locally upon chemoreceptor and pressor receptor elements. The presence of such receptors has recently been demonstrated in the ultimate lung structures.[17]

As in other organs, so in the lungs, hormonal action is most conspicuous in the regulation of its visceral circulation which is quite analogous to that of the liver and kidneys. All these organs have a common vascular hilum and intrinsic vascular nets arranged according to the same general plan. They have separate functional and nutritional blood supplies with anastomoses between these double vascular beds at several levels in the parts and in the units of the organs. The complexity of their vascular nets points to the purposeful functional independence of the organ units where local regulations under central control are mediated by hormonal action as manifestations of the "wisdom of the body."

One manifestation of this has been demonstrated in the visceral circulation of the lungs which is so regulated that blood is constantly diverted to those organ units which at the moment can provide the most effective oxygenation. The mechanism implied in this function involves regulation of peripheral blood flow in the lungs correlated with the momentary levels of gas tensions in the blood of the capillaries and in the air of the alveoli. According to its need for gas exchange, blood is diverted to well ventilated and blocked in underventilated air spaces. The shunting of blood flow from unventilated lungs in so-called N-lung experiments and the remarkable extent to which blood flow has been shown to be diverted from unventilated lung areas even under pathologic conditions[18] are all manifestations of the "wisdom of the body."

Even more remarkably is this demonstrated in the blocking of the flow of fully oxygenated blood to the capillaries of the lungs. This is particularly manifest in congenital cardiac disease which would result in oxygenated blood passing to the lungs. The high pulmonary vascular resistance which develops here was described by Hamilton[19] as "life-saving pathology," for "if a normal peripheral resistance existed in both the systemic and pulmonary vascular body all of the ventricular output would pass out into the pulmonary artery and the pressure in the common ventricle would never rise enough to make blood go out of the aorta and supply the body." While Hamilton referred especially to such congenital cardiac defects as Eisenmenger's syndrome, Tetralogy of Fallot, this applies substantially to all patencies between the two sides of the heart permitting oxygenated blood to pass back to the lungs (left to right shunt). As a matter of fact there is evidence of this phenomenon in congenital cardiac disease with right to left shunts and in pulmonary conditions which involve by-passing of parts of the pulmonary circulation. The latter involve increased blood-flow from the collateral bronchial circulation which being fully oxygenated elicits the same vascular reaction in the pulmonary circulation. Recent observations have demonstrated that the passage of oxygenated blood in the capillaries adversely affects visceral function as indicated by its effect on the diffusing capacity of the lung area involved. The truly functional character of this phenomenon is indicated by its reversible nature. With interruption of the shunt, diffusing capacity returns to normal and upon reopening of the shunt it beomes abnormal again.[20]

Hormonal action upon the capillaries is strongly suggested by observa-

tions made in clubbed fingers. It has been long known that clubbing of fingers develops in pulmonary diseases and in cardiac conditions affecting the pulmonary circulation. It was therefore repeatedly inferred that some substance produced in the affected parts of the lungs is responsible for the phenomena of clubbing. It has recently been demonstrated[21] that clubbing involves structural changes due to altered circulation in the terminal capillaries of the digits. It is reasonable to assume that the stimulus for this altered capillary circulation is derived from the lungs where pathologic changes altering capillary circulation occur at the same time. Furthermore since we know that capillary flow in the periphery of the lungs is regulated locally and its control is mediated by hormonal action, it becomes quite reasonable to consider the possibility that this hormone—possessing specific effect on certain type of capillaries may, by passing from the lungs into the systemic circulation, produce under pathologic conditions clubbing at the body tips, where analogous terminal capillaries exist.

The discussion of the above listed aspects of pulmonary function does not pretend to be complete in any sense. Space does not permit here an exhaustive review of the subject. There are several other aspects of lung function in which much evidence can be gleaned in favor of a special visceral activity, the discussion of which must be left for another time and place. However at least very brief mention should be made here of two more fundamental aspects, namely, "self-cleansing" and "blood depot" function of the lungs. Here again old and recent observations have revealed facts difficult to explain on current views but actually implied in the assumption of visceral lung function.

In "self-cleansing" it has been a long standing problem how inhaled particulate matter reaches the interstitial tissues of the lungs without evidence of having been carried inward by cells lining the air spaces which are supposed to have ingested them.

"Blood depot" function was first postulated by the writer in a publication in 1929.[22] Recent studies have confirmed the fact that the volume of blood normally contained by the lungs is far in excess of their momentary circulation.[23] This raises the great problem not only of the disposition of this large reservoir of blood within the sections of the pulmonary capillary bed but also of its coordination with lung function proper.

In the publication repeatedly referred to above[1] it was shown that the answer to both of these questions lies in the very nature of visceral lung function.

SUMMARY

The concept of visceral lung function was proposed by the writer nearly two decades ago. The problem is here reviewed in the light of some recent developments. The following aspects of lung function and dysfunction are considered.

1) Recent trends indicate that prevailing concepts of lung function are too mechanistic. They have led to the view that elastic recoil, comparable

with that of lifeless rubber, is the sole function of the organ. The need for a more biological concept is obvious.

2) In the establishment of lung function at birth and in the maintainance of the breathing surface throughout life the features of structure and function relationships may reasonably be interpreted as breathing surface creating activity. This is what the writer refers to as the visceral function of the lungs.

3) Recently revealed phenomena of the most purposeful correlation of ventilation and circulation throughout all parts of the lungs, and correlation of diffusing capacity with momentary requirements of the body in health and disease, are not adequately explained by present concepts. These unsolved problems of lung function and dysfunction point logically towards the possibility of an intrinsic function in the lungs, which affords reasonable solutions for these phenomena.

4) The existence of a specific hormone of lung function may be inferred from a number of long known facts and from some more recent observations, which also point to a true organ function of the lungs.

On the basis of the above considerations it is believed that the assumption of visceral lung function is justified at least as a working theory.

RESUMEN

El concepto de la función de los pulmones como órganos fué propuesto hace casi veinte años por el autor. El problema se reexamina aquí, dados ciertos acontecimientos nuevos. Son tratados los siguientes aspectos de la función y de la disfunción de los pulmones:

1) La dirección de las investigaciones recientes indica que los conceptos reinantes de la función de los pulmones son demasiado mecánicos. De ellos proviene la opinión que el rebufo elástico—comparable al del caucho inanimado—es la única función de los pulmones. Se ve claramente la necesidad de un concepto más biológico.

2) El comienzo de la función de los pulmones al nacer consta de la creación de una superficie respirante. Durante la vida, se mantiene esta superficie respirante por la función de las estructuras de las unidades orgánicas fundamentales de los pulmones. Se consideran muchos fenómenos de la correspondencia entre su estructura y su función como ejemplos de la creación activa de una superficie respirante. Esto es lo que llama el autor *la función orgánica de los pulmones*.

3) Los conceptos reinantes no explican adecuadamente ciertos fenómenos, descubiertos recientemente, de la correlación muy a propósito por todas partes de los pulmones, de la ventilación y de la circulación. Asimismo no explican la correlación entre la capacidad de difusión y las exigencias del cuerpo, en el estado de enfermedad como en el de salud, de un momento a otro. Estos problemas no aclarados de la función y de la disfunción de los pulmones sugieren logicamente la posibilidad de que haya una función intrínsica dentro de los pulmones, la cual explica estos fenómenos.

Se infiere de ciertos hechos, conocidos durante mucho tiempo, y de algunas observaciones mas recientes, que hay una hormona especifica rela-

cionada con la función de los pulmones. Estas observaciones también apuntan la verdadera *función orgánica de los pulmones*.

Dadas las consideraciones susodichas, el autor cree que se justifica la suposición de la función orgánica de los pulmones, por lo menos como teoría práctica.

RESUME

Le concept d'une fonction viscèrale du poumon a dèja été proposè par l'auteur il y a près de 20 ans. Le problem est ici reconsidère a la lumière de quelque developments rècentes. Les aspects suivants de la fonction ou de la disfonction du poumon sont considerè.

1) Les tendances rècentes indiquent que les concepts qui prevalent assignent au poumon un role trop mecanique. Elles ont amène le point de vue de la retraction elastique, comparable a celle de caoutchouc inerte, comme seule fonction de l'organe. La necessitè d'une concepte plus biologique est evidente.

2) L'establissement de la fonction du poumon a la naissance, implique le creation du surface respiratoire. A travers tout la vie la surface respiratoire est maintainè par la fonction de structures dernières du poumon. Ici plusiers phenoménes de relation entre la structure et la function prennènt ètre raisonablement interpretée comme activitè de creation de surface respiratoire. C'est dans ce sens que l'anteur parle de fonction viscèrale des poumons.

3) Les faites rècemment revelès de la plus intime correlation entre la ventilation et la circulation à travers toutes les parties du poumon, et la correlation de la capacitè de diffusion gaseux selon les besoins momentanès du corps à l'etat du santè et de maladie, ne sont pas pertinement expliquè par les prèsents conceptes. Ces problèmes non resolus de la fonction du poumon indiquent logiquement qu'il faut rendre vers la possibilitè d'une fonction intrinseque des poumons, qui pourra rendre compte de ces phenomènes.

4) L'existence d'une hormone specifique de la fonction pulmonaire, peut ètre deduite par un nombre de faits connus depuis longtemps et par des observations plus rècentes, qui marquent aussi l'existence d'une veritable fonction d'organe des poumons.

Sur la base des considerations precedents l'auteur sense que l'hypothèse d'une fonction viscèrale du poumon est justifiée, ne serait-ce que comme hypothese d'stude.

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The Diagnosis and Management of Pulmonary Embolism and Infarction*

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Pulmonary embolism and infarction is a relatively common disorder of serious significance which is commonly mistaken for many other pulmonary conditions. Its recognition and treatment should be of great interest to all physicians, particularly those specializing in diseases of the chest. It should be recognized promptly to initiate treatment and to try to prevent recurrences.

Incidence

During recent years there has been a marked apparent increase in frequency which is probably due to an increased alertness of this condition on the part of physicians in general. It was formerly believed that pulmonary embolism and infarction was commonly seen only in patients who had recently undergone operation or delivery. Now we know that it is much more commonly encountered in the practice of internal medicine, particularly in patients with cardiovascular diseases. Although pulmonary embolism has always been common, previously it was diagnosed as all sorts of things, notably pneumonia and pleurisy, and was commonly unrecognized during life. However, during recent years we have come to appreciate its frequency and significance, and the diagnosis can be readily made if the condition is thought of. The incidence is variously reported and is found in about 14 per cent of all carefully performed autopsies.[1] It appears sooner or later in a large percentage of cardiac patients, being found in 48 per cent of one series who died of congestive heart failure.[2] Sixty per cent of all cases occur in medical patients and at least half of these are found in persons with heart disease. Only about 40 per cent occur in surgical and obstetrical patients.[1] A representative figure for a general hospital is about 0.6 per cent of all medical admissions and about 0.25 per cent of all surgical admissions.[3] Pulmonary embolism and infarction account for 5 to 10 per cent of all post-operative deaths.[2] Our experience with patients confined to bed because of pulmonary tuberculosis has shown that this is a relatively infrequent complication.

The sex distribution is about evenly divided between the two sexes. Pulmonary embolism and infarction occur much more commonly in older patients, but is a fairly frequent complication in middle-aged or young adults with heart failure. Eighty-five per cent of the cases occur in persons over 40, and at least half are over 50.[4] It is rare before the age of 20.

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Etiology

The outstanding cause of pulmonary embolism and infarction is a preceding thrombosis of the veins of one or both lower extremities. Careful dissection of the leg veins at autopsy has shown this to be the source in 85 to 95 per cent.[1-4] The thrombotic process usually begins in the plantar or calf veins and then extends up the saphenous and femoral veins. The pelvic, prostatic, and hemorrhoidal veins may be sources of emboli, but not very commonly. Thrombosis of the veins of the upper extremity usually does not result in pulmonary embolism. Even in cardiacs with pulmonary emboli the origin is usually in the leg veins as only 10 to 15 per cent of these patients at autopsy show thrombi in the right chambers of the heart.[5]

Certain patients have a predisposition for thrombo-embolic episodes. These include persons with a history of previous thrombo-embolic episodes; those with extensive varicosities of the lower extremities; patients with congestive heart failure, malignancies, and those undergoing extensive pelvic or abdominal surgery. Pulmonary embolism is twice as common in the obese. Advanced age, particularly over 50, is also a frequent predisposing factor. Trauma to the lower extremities, with and without fracture, and prolonged immobilization also predispose to this complication. Certain hematologic disorders such as polycythemia vera, also predispose to thrombo-embolism.

In the majority of cases there does not appear to be any one precipitating factor, the thrombus breaks loose without any particular provocation and lodges in the pulmonary arteries. However, deep breathing, violent coughing, and straining at stool frequently seem to be the immediate precipitating cause.

Pathology

The structural changes seen in pulmonary embolism and infarction involve three different sites. (1) The thrombosis in the leg vein which becomes the embolus. (2) The actual blockade in the pulmonary circulation by that embolus, and (3) The changes in the lung parenchyma that follow.

The venous thrombus is usually bland and propagates proximally rapidly. It is loosely attached to the wall and its "tail" floats free in the blood stream, free to break loose at any moment. If it becomes fixed and organized it loses its threat and this doubtless happens frequently. When the clot is firmly organized it may produce signs and symptoms of venous obstruction. Until then, local signs are commonly absent. Even when a thrombus becomes fixed there are usually daughter and sister thrombi that are still floating free and may break loose at any time.

The actual blockade of the pulmonary circulation varies in degree and location from almost complete closure of the main pulmonary artery to closure of one of the small branches. Obstruction of one of the main pulmonary vessels may produce death instantly while obstruction of one of the small terminal branches produces minimal or no signs or symptoms. Autopsy studies have shown that the lower lobes are much more frequently involved, about 75 per cent in one series,[1] but involvement of the upper

lobes is unusual. Both lower lobes are frequently involved. Pulmonary emboli are usually multiple, this is particularly true when the emboli are small and recurring frequently.

Pulmonary infarction is by no means a necessary sequel of pulmonary embolism, being found in only one-half to three-fourths of the recognized cases.[1] The pulmonary circulation has rich anastomotic and collateral connections and the development of infarction depends upon four factors: (1) The size of the vessel, (2) The location of the vessel occluded, (3) The presence of pulmonary congestion, and (4) The duration of survival. Obstruction of small or centrally located vessels may not result in infarction because of the rich collateral blood supply. It has been shown experimentally that pulmonary congestion greatly facilitates the production of infarction. The duration of survival is also an important factor as the patient may not live long enough to develop infarction.

It takes several hours for a pulmonary infarct to develop. During the first, and probably the second day, there is only an intense congestion of the capillaries with diapedesis of the red blood cells into the alveoli. There is no definite destruction of alveolar walls. During this stage the infarct is not sharply defined and may simulate focal hemorrhage or bronchopneumonia. After the second day necrosis of the alveolar walls begins and there is degeneration of the red blood cells. This is followed by organization which usually begins during the second week. A fibrous capsule is formed around the area and healing occurs by fibrosis which contracts leaving a linear scar. The infarction may be incomplete and temporary producing only slight or no signs or symptoms and leaving no scar. This is due to the fact that incomplete infarcts heal by resolution rather than organization. Incomplete infarction is usually seen in patients with normal lungs who develop only slight symptoms which last only one to two days and have only indefinite roentgen shadows which disappear in two to four days.

Symptoms

The symptoms of pulmonary embolism and infarction vary widely. They may be entirely absent or be multiple, and vary from mild to overwhelming. Frequently there have been premonitory signs and symptoms of previous small emboli with incomplete infarction. The symptoms produced also depend upon whether embolism progresses to infarction. The most outstanding and commonest symptom produced by embolism is dyspnea which comes on suddenly without warning, is not attributable to effort, excitement, or sudden heart failure, and is out of all proportion to other findings. On rare occasions an asthmatic type of dyspnea is produced. The respiratory distress may be quite brief and may be the only symptom. The next most common symptom is pain which is usually described as a substernal oppression, and in older patients, particularly, may closely simulate angina pectoris. A state of shock is not rare, particularly in the more severe cases or in patients who were already seriously ill. A feeling of faintness, restlessness, and sweating are common complaints. With the development of infarction the patient develops a pleuritic type of pain which usually

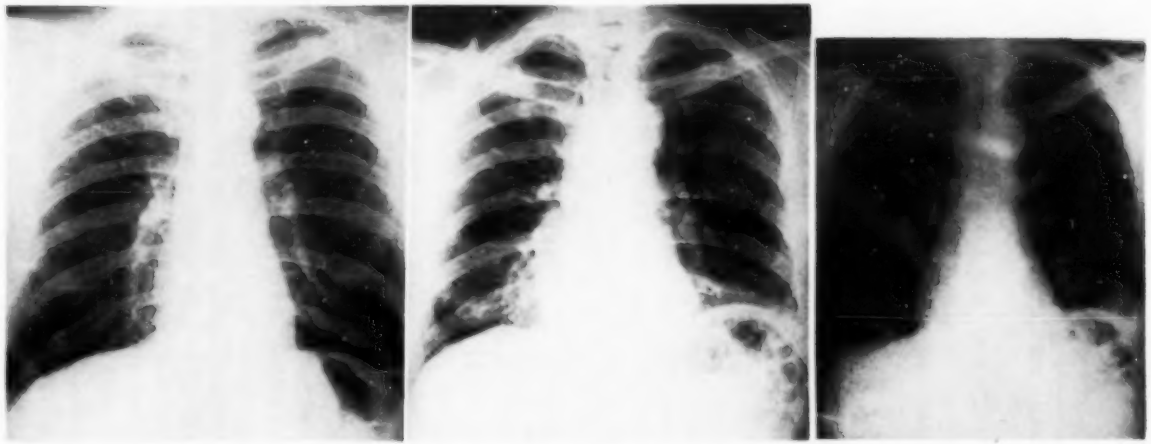

FIGURE 1 FIGURE 2 FIGURE 3

Figure 1: Routine chest x-ray taken June 7, considered normal.—*Figure 2:* Same patient on July 7, one hour after the sudden onset of chest pain and dyspnea. Note the widened mediastinum, prominent right pulmonary vessels and elevated left diaphragm.—*Figure 3:* Forty-eight hours later. Note the indistinct, hazy, round opacity in the left costo-phrenic sulcus.

appears on the second day and persists for several days. Cough is also a common symptom. Hemoptysis is infrequent, being seen in only a third of the cases, but if it occurs, it is important evidence in favor of the diagnosis. It is often quite copious and does not resemble the frothy pink sputum of pulmonary edema.

Signs

Signs may not appear for the first 12 to 24 hours. The most important and most commonly encountered sign is tachycardia which is out of pro-

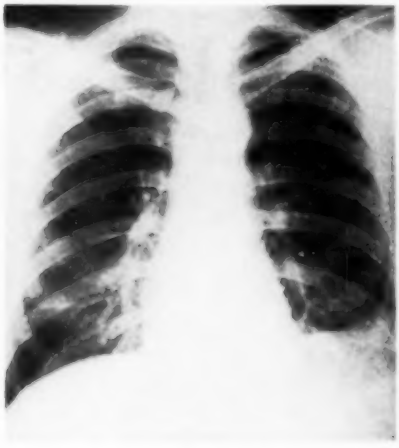

FIGURE 4

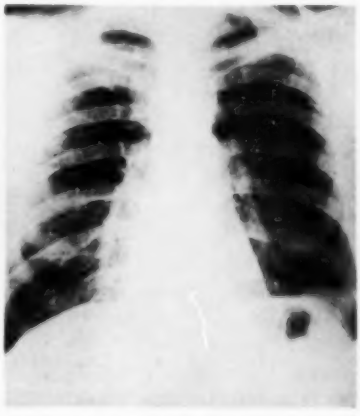

FIGURE 5

Figure 4: Three days later. A left pleural effusion is now present.
Figure 5: Four days later, the infarct is now healing and sharply defined.

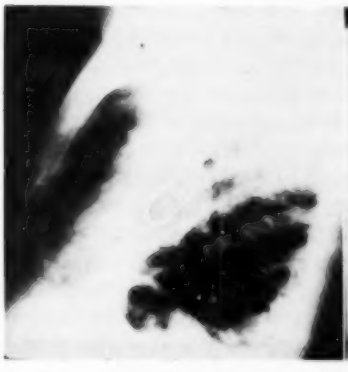

FIGURE 6

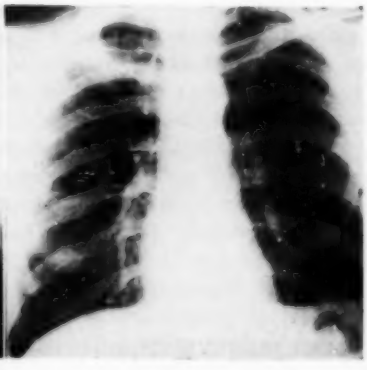

FIGURE 7

Figure 6: Lateral view showing pleural involvement.
Figure 7: Chest x-ray film on August 2. Only linear scarring and irregularity of the diaphragm remain.

portion to all other signs. The tachycardia may be from an ectopic focus but it is usually sinus in origin and often as fast as 150. The next most common sign is that of tachypnea. The third important signs of pulmonary embolism is cyanosis. This is frequently of a high degree and like the other two previous signs is again out of all proportion to the expected findings. Pleural rub may or may not be present, and is less common than the finding of localized, crepitant rales which should be looked for daily. The finding of local signs in the chest aids greatly in the diagnosis. The patient should not be disturbed by asking him to sit up and take deep breaths in an attempt to demonstrate a pleural rub. The movement and deep breathing acts as a sucking mechanism on the great veins of the thorax and may increase the venous pressure four fold in the legs which might precipitate the dislodgement of another thrombus. It is much safer to slip a stethescope under the patient's back and listen during quiet breathing, or better still, slip an x-ray cassette under the patient and taking a roentgenogram. Fever usually develops if there is an infarct or if fever has been present previously, there may be a slight rise. The temperature rise is usually slight and unexplained. If the infarct is massive fever may rise to 103 or 104 degrees F. It should be remembered that the most common cause of fever in a patient with congestive heart failure is pulmonary infarction. Jaundice is an uncommon, but an important confirmatory sign, if present. Thoracic tenderness over the site of the infarct is almost always present and should be sought for. Leg signs, including tenderness, swelling, and Homan's sign are often absent, being found in less than half of the cases,[4,5] but should be sought for daily as their presence aids greatly in facilitating the diagnosis. In addition, there are usually the signs of obstruction of the pulmonary circulation which include an accentuated pulmonic second sound, venous distention, slight hepatomegaly, and a systolic murmur and/or a friction rub heard over the pulmonary area.

Laboratory Data

The signs and symptoms enumerated above are much more important in making the diagnosis, but sometimes the laboratory is a distinct aid in pointing to the correct diagnosis or in confirming the clinical impression. The roentgen ray examination is the most valuable of all laboratory procedures. At the onset and for 24 hours or more there may be little to find wrong except slight elevation of the diaphragm on the affected side together with distended main pulmonary trunks with decreased caliber of the pulmonary vessels distally.[1,6] These are helpful signs which should always be looked for. It is to be emphasized that a negative chest x-ray film, particularly one obtained during the first two days, does not eliminate the diagnosis of pulmonary embolism or infarction. If the process does not progress to infarction there may be no additional x-ray findings. If infarction ensues, one or more areas of increased densities appear. These are usually in the lower lobes, at first are hazy and poorly defined with indistinct margins. As necrosis is followed by organization and healing the infarct becomes distinct and sharply defined. The lesion is always

peripheral with one or more pleural surfaces involved, usually at their junction. They are rarely triangular, are usually round, oval, or irregular. The cardiac surface is convex or "humped". If triangular, the apex is peripheral, the long axis of the infarct parallels the long axis of the pleural surface involved. Pleural effusion occurs in about half of the cases and is usually a bloody exudate. The infarct may be obscured by the effusion, or by an enlarged heart, or may be buried in the costophrenic angles. With incomplete infarction the shadows last only two to three days and heal without residual scarring.

The electrocardiogram is of no assistance in at least half of the cases. It is more likely to be of help if taken early, if the infarct is large, and in older patients. The pattern is characteristic and is usually described as being due to right ventricular dilatation and consists of a prominent S wave in lead I, a Q wave often appears in lead III, the T wave is usually inverted in lead III and in the right precordial leads. There may be considerable elevation of the RST segments over the right side of the precordium with reciprocal depression over the left side. The transition zone is commonly displaced to the left. Transient right bundle branch block, either complete or incomplete, may appear. Arrhythmias are common, atrial fibrillation or flutter may be seen in as many as one-third of the cases and is usually paroxysmal. Tachycardia, usually sinus, is almost always present. Other laboratory findings consist of slight to moderate leukocytosis which is usually present and is due to the infarction. The sedimentation rate is elevated and the serum bilirubin may be slightly elevated.

Course and Prognosis

Pulmonary embolism may kill very quickly within a matter of a few minutes but more often recovery ensues. Only a few die within a few minutes, a few more live for about one hour, but two-thirds live several hours to several days. It has been stated[7] that 25 per cent die of their first attack but this figure is probably too high as many mild cases are never recognized. Of the cases that survive, 30 to 40 per cent suffer recurrences which is one of the outstanding characteristics of the disorder. One large series[7] showed that 80 per cent of the recurrences occurred within 10 days of the initial insult. Like the original attack, the severity varies widely. Pulmonary embolism and infarction may occur at anytime within three months of operation or injury, but the most common time is during the second week. About one-half of all cases occur during this period, one-fourth during the first week, and the remaining one-fourth after the 14th day. Recovered cases may show no sequelae whatsoever or they may show linear scars on roentgen ray or autopsy. Some cases of massive pulmonary embolism may recover to slowly develop a chronic cor pulmonale due to the obstruction in one of the main pulmonary vessels which results in marked pulmonary hypertension. Repeated episodes of embolization in the tertiary radicals of the pulmonary arterial tree may also result in pulmonary hypertension and chronic cor pulmonale.

Complications

The commonest complication of pulmonary embolism is pulmonary infarction which has already been described. Acute vascular shock is a frequent and serious complication particularly in the aged and debilitated. Another complication is myocardial infarction which is probably due to the combination of previously diseased coronary arteries and anoxia but has been attributed to reflex coronary spasm mediated by way of the vagus nerve. A third complication is the development or worsening of congestive heart failure. It is a good rule to suspect pulmonary infarction in any patient who develops or shows worsening congestive heart failure without an obvious cause.

Differential Diagnosis

The conditions which are most frequently confused with pulmonary embolism and infarction are pneumonia, myocardial infarction, congestive heart failure and pleurisy. Sometimes spontaneous pneumothorax or mediastinal emphysema may be confusing. The differentiation is usually easy by history and physical examination alone, but now and again the chest x-ray film or electrocardiogram may solve the problem. The most important point in the differential diagnosis is the course of the illness with recurrent attacks. The frequent recurrence of pulmonary embolism is one of its chief diagnostic features and distinguishes it from the two conditions with which it is so often clinically confused; namely, myocardial infarction and pneumonia. Neither one of these conditions recur rapidly every few days. A patient may have acute coronary occlusion or pneumonia initially, but when some acute complication in the chest keeps repeating itself, one should think of pulmonary embolism and suspect that even the first attack may have been that condition. Pneumonia does not tend to recur repeatedly nor does myocardial infarction do so, particularly in a patient who survives. In addition to the recurrent feature, other important clues which should point toward the correct diagnosis are a history of recent operation or injury, particularly to the lower extremities; an unexplained rise in temperature, pulse, and respiration, particularly in a bed patient; the abrupt onset of dyspnea, tachycardia and tachypnea out of proportion to the temperature; the finding of thoracic tenderness, or leg signs, or unusual cyanosis. Hemoptysis of any degree, particularly frank bleeding, should always point to pulmonary infarction as this sign is a rare manifestation of pneumonia and is not encountered in uncomplicated pleurisy or myocardial infarction. Any patient who shows the sudden onset or sudden worsening of congestive heart failure should be suspected of having either a pulmonary or myocardial infarction. Remember, the most common cause of fever and/or jaundice in a patient with congestive heart failure is pulmonary infarction.

Treatment

Slight cases of pulmonary embolism may require no treatment except prophylactic, but cases of severe degree need emergency and often heroic

treatment. The first few hours are critical and the emergency treatment should consist of either morphine or demerol, oxygen by tent or mask in high concentrations, and expert nursing care. The patient should be at absolute bedrest and instructed to avoid deep breathing, violent coughing and straining at stool in hopes of preventing another attack. Unless there is some contraindication anticoagulant therapy should be initiated immediately and this should consist of heparin and dicoumerol or tromexan. The heparin should be continued until the prothrombin time has reached a therapeutic level which is two to two and one-half times the control. Anticoagulant therapy should be continued until the patient is fully ambulatory and should be continued indefinitely for cases showing repeated insults. The leg veins should be investigated and if there are multiple emboli bilateral femoral vein ligation should be carried out. It is to be emphasized that venous ligation should be combined with anticoagulant therapy as it has been shown that venous ligation alone cannot be relied upon. Thrombus formation has been found proximal to the site of ligation in over half of the cases studied at autopsy.[3] It is doubtful if the inferior vena cava should ever be ligated and then only as a last resort. If shock is present it must be combatted and probably the most effective agents are the vaso pressors such as the sympathomimetic amines and norepinephrine. Venous transfusions should be avoided as the venous system is already congested and distended. Probably arterial transfusions would be of value. Although venous congestion is present it is wise to avoid venesection because of the ever present danger of shock which might be precipitated by a depletion of blood volume. Atropine and papaverine are of little value. Antibiotics should be given prophylactically.

The prevention of venous thrombosis in the leg veins is more important than its treatment. At the present time it is not definitely known which is the most effective prophylactic treatment. Certainly, a state of physical and circulatory fitness should be established and maintained, especially prior to confining a patient to bed. Any position or procedure conducive to blocking of the pelvic and leg veins should be avoided. Leg circulation should be fostered by passive and active exercises and getting the patient out of bed at the earliest possible time. Recent reports[4,5,8,9] have shown that the early ambulation of surgical patients has failed to materially reduce the incidence of fatal pulmonary embolism and infarction. This may be more apparent than real because in recent years, with the advent of antibiotics, better anesthesia, and more liberal use of blood, many older patients are now undergoing extensive surgery. Perhaps there would have been a distinct rise in the incidence of fatal pulmonary embolism and infarction in these patients if early ambulation had not been practised. The routine venous ligation and post-operative use of anticoagulants is open to question; however, all cases prone to thrombo-embolic episodes should be so treated. It is well to remember that once a thrombo-embolic, always a thrombo-embolic. Recently it has been shown in one clinic[10] that the wearing of knee length, elastic stockings has some value in the reduction of pulmonary embolism and infarction.

SUMMARY

1) Pulmonary emboli and infarction are common, serious complications in patients confined to bed for one to three weeks by an operation, fracture, delivery, or medical illness.

2) Pulmonary infarction is not an inevitable sequel of pulmonary embolization.

3) The signs and symptoms vary widely.

4) The diagnosis is usually easy, but is frequently missed. The unforgivable sin is not in missing the diagnosis but in not considering it.

RESUMEN

1) Las embolias pulmonares y los infartos son comunes y serias complicaciones en los enfermos mantenidos en cama por una a tres semanas con motivo de una operación, fractura, parto o enfermedad no quirúrgica.

2) El infarto pulmonar no es una inevitable secuela de la embolia pulmonar.

3) Los signos y los síntomas varian ampliamente.

4) El diagnóstico es habitualmente facil pero a menudo no se hace. El pecado imperdonable no esta en no hacer el diagnóstico sino en no pensar en esa posibilidad.

RESUME

1) Les embolies et les infarctus pulmonaires sont des complications sérieuses et fréquentes chez les malades confinés au lit pour deux ou trois semaines, par une opération, qu'il s'agisse de fracture, d'accouchement ou d'affections médicales.

2) L'infarctus pulmonaire n'est pas la conséquence obligatoire de l'embolie pulmonaire.

3) Les signes et les symptômes sont extrêmement variables.

4) Le diagnostic est habituellement facile, mais il est souvent non fait. Ce n'est pas se tromper de diagnostic qui est la faute impardonnable, mais c'est de n'avoir pas pensé à le discuter.

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So-Called Hypertrophic Emphysema

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Before discussing the pathogenesis and clinical aspects of this disease, clarification of some of the pertinent terminological definitions may be in order. Hypertrophic emphysema is the most commonly used designation of this disease. Also, it is known under the following names: genuine emphysema, idiopathic emphysema, diffuse vesicular emphysema, obstructive emphysema, dystrophic emphysema, pulmonary hypertrophy, pulmonary dilatation, chronic large lung, and pneumonectasis. Inasmuch as loss of the elastic fibers and more or less extensive destruction of the alveoli are cardinal aspects of this disease, one may designate it as destructive emphysema. Still a more appropriate term, pseudohypertrophic emphysema, may be offered for the nomenclature of this condition. Hypertrophy of the lung in genuine emphysema is far from being similar to muscular hypertrophy of the extremities seen in men engaged in athletics or in occupations which entail heavy physical work. Although in genuine emphysema the lung is larger than normal, there is neither an increase in the number of functionally competent alveoli nor a proportionate augmentation in the number, size and functional capacity of corresponding capillary vessels.

During the course of development of genuine emphysema a great many interalveolar septums are destroyed. Consequently, large, cyst-like air spaces appear in the lung. Cyst-like formations of this type are designated as blebs when they are localized subpleurally. The ones localized in other parts of the lung are conventionally referred to as bullae.

Concepts vary relative to the pathogenesis of genuine emphysema. I am of the opinion that the following factors are of cardinal importance in this respect: (1) Increased intrapulmonary pressure during strenuous coughing. (2) Infections and other pathologic changes which result in extensive degenerative alterations in the elastic elements of the lung. (3) Partial bronchial occlusion of the check-valve type. Familiarity with the role of each of these items in the pathomechanics of genuine emphysema is helpful in the satisfactory management of this disease.

During the compressive phase of cough which is strenuous, the intrapulmonary pressure may be as high as from 80 to 200 mm. of mercury over and above atmospheric pressure. This aerodynamic trauma, when sustained for a prolonged period of time, is likely unduly to stretch and to damage the elastic fibers of the lung.

Chronic lung infections may lead to destructive changes in the elastic elements of the lung in two ways: (1) through direct toxic influences; (2) through consequent fibrosis. The latter causes constriction or complete obliteration of some of the nutritional blood vessels. The consequent diminished blood flow exerts an untoward influence upon the implicated lung

structures. Similar sequels may follow prolonged, massive inhalation of noxious fumes, gases and dusts.

Widespread spasm of the peribronchial and peribronchiolar smooth muscles is of more common occurrence than generally realized. Its presence in allergic bronchial asthma is well known. Also, it may be provoked by infections of the lower respiratory tract, by inflammatory hyperemia, by congestion due to failure of the left ventricle and by extensive pulmonary fibrosis. Bronchospasm, particularly in the presence of congestion and edema of the bronchial mucosa and instances where inflammatory exudate narrows the bronchial lumen, is likely to set up a check-valve mechanism. The latter permits the ingress of air to parts of the lung distal to the partial occlusion. Inasmuch as the egress of the entrapped air is prevented, there develops in widespread areas of the lung what may appropriately be called pneumatic alveolar hypertension.

The deleterious effect of pneumatic alveolar hypertension can be more readily appreciated if the following changes are borne in mind. (1) Air currents are subject to the same physical laws as water currents. They move from a site of higher pressure toward areas of lower pressure. While during quiet respiration the intraalveolar pressure cannot be higher than the atmospheric pressure which prevails in the bronchi, during the compressive phase of strenuous coughing the intraalveolar pressure may rise to 200 mm. of mercury over and above atmospheric pressure. (2) The physical law of communicating vessels applies to the relationship between respiratory bronchioles and their respective cluster of alveoli. The same pressure which exists in the narrow respiratory bronchiole is transmitted undiminished to the entire perimetry of all of the respective alveoli. (3) At the termination of the compressive phase of cough, when there is a precipitous drop in the intrapulmonary pressure, evacuation of the air is slower from alveoli attached to spastic, partially occluded bronchi than from alveoli connected to bronchi of normal lumen. This exerts an appreciable distending influence upon the alveoli implicated.

The normal configuration and size of the thorax are dependent on anatomic as well as on physiologic factors. The physiologically stretched out position of the lung is maintained (1) by the pressure of the atmospheric air; (2) by the centrifugal suction effect of the intrapleural negative pressure. The thoracic cage is larger than the lung. Even so, the lung is held in apposition to the inner surface of the chest wall by the traction of the intrapleural negative pressure. The latter is an expression of the difference between the pressure of the atmospheric air reaching the lung through the lower air passages and the innate centripetal (hilusward) retractility of the elastic elements of this organ.

When as the result of loss of the elastic elements of the lung, its centripetal (hilusward) contractility is greatly reduced, there is a proportionate decrease in or complete disappearance of the negativity of the intrapleural pressure. The inspiratory muscles of the chest wall, not being obliged to counteract the inward pull of the intrapleural negative pressure, are bound to distend the thoracic cage. This train of events is similar to that seen

when initial artificial pneumothorax is given. It is obvious, therefore, that distention of the thorax as well as the enlargement of the lung in genuine emphysema are brought about by the same factor, namely by the loss of the elastic contractility of the lung.

The normal position of the diaphragm is the direct result of the upward traction force of the intrapleural negative pressure. In genuine emphysema, in consequence of the disappearance of the upward traction of the intrapleural pressure, the diaphragm occupies a constant low (inspiratory) position. Clinically, it is easily demonstrable that the diaphragm occupies a low position after the establishment of artificial pneumothorax. Also, it is known that the respiratory excursions of the diaphragm are restricted following the institution of artificial pneumothorax. The reason is that in an abnormally low position the diaphragm is functionally handicapped or completely defunctionalized. In emphysema, its respiratory motions are slight, absent or may be paradoxical: rising on inspiration and descending on expiration.

In addition to impaired diaphragmatic function, the following functional changes are noted in so-called hypertrophic emphysema: (1) Lessened negativity of the intrapleural pressure. (2) Decreased respiratory excursions of the ribs. (3) Diminished return of venous blood to the right auricle because of the less negative intrathoracic pressure. Consequently, the venous pressure is elevated. (4) Decrease in or lack of dilatation of the pulmonary vascular bed on inspiration. (5) Pulmonary hypertension. (6) The pulmonary tidal air is normal or slightly reduced. (7) The complemental air is reduced. (8) The functional residual air is from two to three times that of normal. (9) The maximum breathing capacity is reduced. The reduction may be as much as 50 per cent or more. (10) The vital capacity of the lung may be lowered by from 20 to 60 per cent. (11) Carbon dioxide content of the alveoli is increased to 7-8 per cent (50-60 mm. of mercury). (12) Oxygen saturation of the blood is below normal. It may be as low as 60 per cent. (13) There is an increase in the bicarbonate reserve in the blood. (14) Plasma chlorides are decreased. (15) Polycythemia is present without pathologic bone marrow changes. (16) The size of the erythrocytes is increased.

So as to avoid misguided therapeutic intervention, I wish to present the salient differential diagnostic aspects of genuine emphysema and senile emphysema. The term emphysema is derived from the Greek words which mean "in" and "to blow". Applied to the lung, this term signifies inflated lung. According to the derivation of this term, senile emphysema is a misnomer, for in this condition the lung is small, with the exception of the Kountz-Alexander type of senile emphysema. On postmortem examination when the chest is opened, the lung collapses in senile emphysema. Some of the alveoli are dilated in this condition. In the aged, the alveoli become flabby like the skin. The consequent dilatation of the alveoli is the result of atrophic changes in the alveolar septums, corresponding to the age of the individual. Similar degenerative changes prevail in all of the senescent body tissues. Simultaneously, some of the elasticity of the

lung is lost. But it is to be kept in mind that this loss is proportionate to the loss of the tone and strength of the respiratory muscles of the chest. Consequently, the normal functional balance remains undisturbed between the centripetal (hilusward) traction of the pulmonary elastic fibers and the centrifugal traction of the intrapleural negative pressure. The latter is actuated by the outward pull of the inspiratory muscles of the chest

DIFFERENTIAL DIAGNOSIS

	Pseudonypertrophic Emphysema Increased	Senile Emphysema Not Increased*
Size of the Chest		
Shape of the chest	Barrel-like or box-like	Small, flat*
Position of ribs	Horizontal	Oblique*
Position of sternum	Elevated	Not elevated*
Intercostal spaces	Widened	Narrowed*
Dorsal spine	Kyphosis	Straight or convex
Neck	Short	Normal*
Shoulders	Thrown forward	Normal*
Accessory respiratory muscles	Visible function	No hyperfunction
Epigastric angle	Widened	Normal or narrowed
Motion of epigastrium	Not protruding on inspir.	Inspir. protrusion
Pulsation of epigastrium	Present	Absent
Motion of lower ribs	Inward on inspiration	Normal
Diaphragm: position	Low	Normal
Diaphragm: excursions	Decreased or absent	Normal
Type of respiration	Thoracic	Abdominal
Cyanosis	Present	Infrequent or absent
X-ray: Costophrenic sinus	Shallow	Normal*
X-ray: Translucency	Increased	Normal or slight incr.
X-ray: Lung behind sternum	Increased	Not increased*
X-ray: Vascular markings	Indistinct	Normal or increased
X-ray: Shape of sternum	Anterior convexity	Normal
X-ray: A-P diameter of chest	Increased	Not increased*
Pulmonary function	Insufficiency	Normal
Arterial oxygen saturation	Low	Normal
Arterial CO_2 tension	High	Normal
Signs of right heart failure	Often present	Absent
Venous pressure	Increased	Normal

*Except in the Kountz-Alexander type of emphysema.

wall. In senile emphysema the respiratory excursions of the chest are reduced also because in the aged there are calcification of the costochondral junction, limitation in motion of the costovertebral joints, fibrosis and loss of elasticity of the thoracic ligaments, loss of elasticity of the bronchi due to calcification of their walls, possible pulmonary arteriosclerosis and concurrent heart failure. Pulmonary congestion associated with heart failure causes a decrease in the expansability of the lung.

The Kountz-Alexander type of emphysemas, according to the original definition of these clinicians, is not primarily a pulmonary disease. Changes in the lung are secondary to an increase in the size of the thoracic cage. One of the characteristic features of this condition is straightening and stiffness of the thoracic spine. These changes are brought about by degenerative alterations in the intervertebral disks. The nucleus or the entire disk becomes swollen. In the advanced degenerative phases some of the intervertebral disks become completely separated from the bone and undergo complete dissolution. With the progression of degenerative changes, the corresponding vertebral bodies become implicated, they thin out and finally, kyphosis may result. This, in turn, will lead to an outward flare of the ribs and to the development of barrel chest. The lung passively follows the distention of the thorax and thus it becomes somewhat enlarged in volume.

Treatment

It is almost a platitude to say that we as a nation are growing older. Since the turn of the century the population of the United States doubled, while the number of aged quadrupled. Today 8 per cent of the population, that is, more than 12 million out of 156 million, are over the age of 65. With this constantly lengthening life span, it is anticipated that the number of persons over 65 years of age will reach more than 13 per cent of the population of this country at the end of this century. All this being so and also, bearing in mind the pathogenesis of so-called hypertrophic emphysema, more and more instances of this disease are bound to come to the attention of practicing physicians.

Since mass x-ray surveys, preemployment x-ray examinations of the chest and chest roentgenograms on admission to general hospitals are accepted by the medical profession as well as by the public, it is justifiable to point out the possible recognition of subclinical forms of this disease. With its early detection, appropriate measures may be instituted for obviating its progression. At the same time, development of complications may be prevented.

In my personal experience, I have found artificial pneumoperitoneum a highly satisfactory method in the management of so-called hypertrophic emphysema. Technical details of this procedure are presented in the author's monograph, "Pneumoperitoneum Treatment". In addition, four points deserve particular emphasis.

1) The amount of air given with each treatment should be less than 1,000 cc. Larger amounts of air are bound to cause limitation in motion of the diaphragm. Thus the treatment would defeat its own purpose.

2) Pneumoperitoneum treatment can be started in the office or in the clinic. "Refills" are given at weekly intervals.

3) The patient should wear a snuggly fitting abdominal girdle night and day. Elastic segments of the girdle should be small. Metal plates, padding or inflatable compartments are not essential. The girdle should not interfere with the respiratory motion of the lower ribs.

4) Artificial pneumoperitoneum is well tolerated by the patient, provided no extensive peritoneal adhesions are present. Treatments may be continued for years.

The following factors are responsible for the beneficial results of artificial pneumoperitoneum:

1) Artificial elevation of the diaphragm refunctionalizes this previously defunctionalized muscle.

2) Pneumoperitoneum emancipates the diaphragm from the gravitational pull of the liver and of the spleen.

3) Increased intraperitoneal pressure caused by pneumoperitoneum is associated with reflex relaxation of the peribronchial spastic smooth muscles.

4) In so-called hypertrophic emphysema, artificial pneumoperitoneum renders the intrapleural pressure more negative. This greater negativity draws the diaphragm upward and the chest wall inward. Thus the function of the corresponding inspiratory muscles is improved.

5) Pulmonary pneumatic dyskinesia (faulty distribution of the inhaled air) is corrected. Consequently, the oxygen-carbon dioxide exchange is improved.

6) Oxygen saturation of the arterial blood is increased and its carbon dioxide concentration is decreased.

7) The increased negativity of the intrathoracic pressure facilitates the return of venous blood from the periphery of the greater circulation to the heart, with consequent decrease in the venous pressure.

8) There is increased blood flow from the heart to the lung. This relieves strain on the right ventricle.

9) Cough mechanism is improved.

While my own experience as well as the findings of others offer convincing evidence of the efficacy of pneumoperitoneum in the management of so-called hypertrophic emphysema, this treatment should not be looked upon as a cure-all. Artificial pneumoperitoneum is of limited value or of no value at all in the following instances:

1) When there is anatomically and functionally irriversible, extensive loss of alveoli and elastic elements of the lung.

2) When adhesions prevent the elevation of the diaphragm.

3) When there is atrophy of disuse of the diaphragm in long standing emphysema.

4) In cases of heart failure which cannot be corrected.

5) When uncontrollable complications interfere with cardio-respiratory function.

While I advocate artificial pneumoperitoneum as the method of choice

for the treatment of genuine emphysema, I do recommend, whenever circumstances so require, the use of antimicrobial drugs, chemotherapeutic agents, bronchorelaxant medicaments, hyposensitization, the administration of digitalis preparations and diuretics and other forms of supportive and specific treatment.

For completeness' sake, I wish to mention other methods which have been advocated for the treatment of this condition.

1) Expiratory pressure breathing with pursed lips was first recommended by Schutz as a purposeful therapeutic measure in 1935.

2) Oxygen in gradually increased concentrations given by inhalation was first advocated by Barach in 1938.

3) Intermittent positive pressure breathing of oxygen was first introduced by Motley and his associates.

4) Manual compression of the lower anterior part of the chest, the upper part of the abdomen or both, rhythmically at intervals corresponding to the expiratory phase of the respiratory cycle, has been advocated by clinicians in this country and abroad during the past few decades.

SUMMARY

A review of the pathomechanics of so-called hypertrophic emphysema qualifies artificial pneumoperitoneum as a logical, preferential method of treatment of this disease.

RESUMEN

Una revisión de la mecánica patológica del llamado enfisema hipertrófico permite calificar al neumoperitoneo artificial como el método de elección para el tratamiento de esta enfermedad.

RESUME

L'auteur, après avoir passé en revue les facteurs physio-pathologiques de l'emphysème que l'on qualifie d'hypertrophique, montrent qu'ils permettent de considérer le pneumopéritoine comme la méthode de traitement logique et supérieure à toute autre.

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Comments on Detection of Cardiac Disease in Group Examinations*

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In this brief presentation remarks will be confined to the discovery and evaluation of heart disease as found in pre-employment examinations, in college entrance examinations, and in periodic re-examinations. Although the zeal for disease control may not exist for heart as it does for pulmonary disease, it is equally challenging in preventive medicine to attempt to discover and control latent heart disease before it assumes its devastating and incapacitating stages. Prompt recognition of heart disease has important implications for the victim's welfare; by the same token early assurance that suspected heart disease is not present has important significance for any person and particularly for the individual who is subject to unfounded fear. Care should be taken that the patient is promptly advised by his physician concerning significant abnormality. In personal experience, the patient with cardiac disease who has received a constructive explanation of his defect usually feels and ofttimes expresses appreciation.

The person who, on his own volition, seeks aid from the physician for the relief of symptoms or for health information is the individual who will be the most cooperative and most receptive of health advice. The practicing physician in his routine work has an unequalled opportunity to teach health habits and to discuss prevention as well as treatment of disease. It is advisable for the patient's own physician to present to him the findings of the examination. A certain degree of normal anxiety prompted the medical consultation; therefore, sound advice on the part of the physician should orient the individual with regard to his complaint. Those who come from physical surveys, pre-employment and school entrance examination, need especially to be appraised of the value of the findings of the examinations and their follow-up. At the other end of the clinical spectrum of patients with excessive anxiety stand those with indifference and sometimes boredom. By and large, examination of healthy young people instills good medical habits and teaches the coming generation to seek and evaluate advice from their physicians. It also may be said that, more and more, industry is attempting to recognize latent disease and defects by offering pre-employment and periodic examinations with the attempt to adjust work requirement to the individual's physical capacity.

History taking is an important part of the examination and is helpful if accurate and to the point. The history of rheumatic heart disease taken years after an attack in childhood may be unreliable but in all cases in question should lead to a careful examination. But once the examination

*Presented before the Medical Staff of the Student Health Service, University of Minnesota, March 2, 1953.

is negative and definitive, one should not hesitate to tell these individuals that they are normal even in the presence of a supposedly positive history. Too many people who have recovered from any demonstrable residuals of rheumatic fever have been left in a quandary with regard to the existence of heart disease. However, it must be kept in mind that sometimes signs of rheumatic heart disease are delayed for some years after the acute attack.

With regard to degenerative (arteriosclerotic) heart disease much more emphasis may be played upon a careful and accurate history. When an individual in the fourth decade and beyond has a definite, fairly abrupt diminution in his physical capacity—that is, has become short of breath quantitatively for tasks previously performed with ease—a positive indication of impaired cardiac function is present. Further study must indicate whether such impairment is due to intrinsic cardiac cause or extrinsic cause such as anemia. For instance, cases of leukemia often appear with dyspnea due to anemia as the chief complaint.

The recognition of the insidious onset of cardiac disease at times is important. The unrelenting fatigue of the 55-year-old auto mechanic (O. A.), examined over a period of years, was a true sign of developing coronary arteriosclerosis and impending fatal thrombosis, even though all other findings were negative except for low voltage of QRS complexes and they were interpreted to be within normal limits. In certain instances patients have admitted that the first sign of coronary insufficiency was shown when the effort of walking a few blocks would provoke substernal distress enough to prompt them to stop and windowshop on the sidewalk until relief was obtained. This is personally called "the window shopping sign" of coronary insufficiency. Interpretation and analysis of the history becomes the most significant feature of the examination.

With regard to the physical examination, the occurrence of sinus tachycardia will first be mentioned. This most common cardiovascular abnormality often has no significance as to disease but still has significance in the total estimation of the individual. This may be called an "examination tachycardia," and there may be no other abnormal findings. Patients with so-called neurocirculatory asthenia or effort syndrome may show reactive tachycardia. Some of these individuals assert, and it is hoped accurately, that their pulse rate usually is in the 70's or 80's. A recent individual's (M. B.) lowest pulse rate ever recorded by numerous observers was 110. His usual rates were 120 and over. All phases of his physical and laboratory examinations were negative. At times these individuals relate that they are scared only when in a doctor's office. One is inclined to say that they probably respond to many life situations with tachycardia from loss of vagus tone or from sympathetic stimulation. With distraction of the patient, occasionally the rate will slow during the period of the examination. Assurance to these people is essential without too frequent recheck examination.

More difficult of explanation is the group that show, silently and as an isolated finding, transient or labile systolic and disatolic hypertension

without tachycardia. While it may be easy to put these individuals in the general category of labile hypertension, determination of the irritative provocation is worth seeking. In some instances, stressful living and school conditions appeared to be provocative. Since such cases of hypertension are picked up by chance without other associated findings, a record of sustained elevation of pressure should probably be obtained before reporting any single variation to the patient. Most physicians have to adopt and use the method of frank explanation, but also realize that not all variations need be analyzed in detail before the patient.

The paroxysmal tachycardias are an important group of cases whose early recognition will save the individual needless anxiety or incapacity that might arise from inadequate or mistaken diagnosis of serious heart disease. Often this disturbance of rhythm is an isolated finding in a normal heart. The history is usually quite definitive and reveals in an individual without other complaints episodes of abrupt onset of racing or pounding of the heart, lasting usually for a few seconds or minutes; extreme cases may last for hours. Breathlessness with slight precordial discomfort may occur with the attack. Usually no provocation is apparent. Confirmation of the diagnosis is made by observation of an attack and by electrocardiogram to determine whether auricular, ventricular, or nodal in origin. In cases followed for some years, and in one instance (Mrs. S. E.) followed for over two decades, the attacks have become less frequent and the heart has remained normal throughout.

Just as important at times is the recognition of arrhythmia due to premature contractions. This most common type of arrhythmia in young and normal individuals may become, if not correctly interpreted, the basis for psychogenic heart disturbance. Careful study reveals the occurrence of a "turn over" of irregularity or sometimes of a pounding of the heart with or without tachycardia. The finding of extrasystoles and the determination of their frequency, followed by determination of normality of the heart, form the basis of rational procedure and instruction of the patient, with uprooting of cardiac fears. It should be mentioned that sometimes in arteriosclerotic heart disease the occurrence of increased frequency of ventricular premature contractions may be a precursor of increasing myocardial irritability and possible damage, with total arrhythmia. By and large, the isolated finding of premature contractions has no clinical significance. In the normal individual, this irregularity may lessen with tachycardia following exercise.

The detection of cardiac murmurs, with their interpretation, becomes a common source of screening cases of cardiac disease through routine physical examinations. Physicians have been intrigued by sounds in the chest since the discovery of auscultation by Laenec. The stethoscope is available to physician and surgeon alike, and its use is deterred by nothing except lack of interest or inability to interpret what is heard. The subject of cardiac auscultation is broad and worthy of more detailed study, but only a few angles of the problem will be mentioned here.

The isolated findings of a grade one or two systolic murmur at the base

of the heart, usually over the pulmonic area, need not require further examination or reference and may be accepted as functional or physiologic. It is present in a considerable number of individuals if adequate time is taken to ferret it out. No useful purpose is served by further recheck unless the examiner desires study concerning other questionable cardiac findings. The pulmonic functional murmur is associated with normal dynamics rather than derangement of structure. Conditions which increase the velocity of blood flow, such as anemia, pregnancy, hyperthyroidism, and febrile states, are commonly associated with these functional or, better yet, physiologic murmurs.

The systolic murmur becomes of more significance if it is loud, grade three to four, and prolonged; if it is apical; if it is transmitted beyond the cardiac border; if it is constant; if it is not variable with respiration and posture; or if it is associated with marked variation in intensity of cardiac tones, especially with masking of the first sound at the apex in mitral insufficiancy and accentuation of M_1 and sometimes P_2 in mitral stenosis; also if it is associated with a distinct thrill or with cardiac hypertrophy. The detection of such murmurs is often the first step in establishing structural change in the mitral valve with insufficiency and with stenosis if the presystolic and mid-disatolic murmurs appear.

Moreover, conditions that cause dilatation of the heart may cause dilatation of the annulus of the mitral valve whose leaflets and chorda tendineae themselves are normal. In the records of the University of Minnesota Hospital 32 years ago, is the report of the case of a young woman with exophthalmic goiter who developed postoperative thyroid crisis and in whom, overnight, a loud intense systolic murmur appeared, only to disappear almost as rapidly within one or two days as her recovery progressed. Due to similar dilatation is the appearance of systolic murmurs in the failing heart, such as may occur in myocardial infarction. Loud vibratory murmurs have appeared, due to ruptured leaflets, cusps or chorda tendineae. In M. D., a fatal case of calcareous mitral stenosis with small fish-mouth aperture, the paradox has been observed that the thrill and murmur tended to disappear completely as the cardiac output lessened with failure.

Categorically, it may be stated that the prolonged diastolic murmur with or without associated cardiac findings is considered evidence of aortic insufficiency. The degree of left ventricular strain present in some of these aortic cases is inadequate to cause contour changes until after years. It is also true that the peripheral vascular signs of Corrigan and Duroziez may be likewise delayed in appearance.

A practical point in examination of large groups is that the individual who is asked to come back for further cardiac examination is sometimes unduly alarmed. This is also true in the recheck case in which the patient is told to have an electrocardiogram with contour study and possibly Master two-step test done before cardiac consultation. A word of explanation will often obviate an anxiety reaction, for sometimes the first step in iatrogenic heart disease is occasioned by the words or action of the phys-

ician who has not taken time to explain, after a competent examination, that this or that supplemental procedure was for the purpose of thoroughness. Minor signs found by that examination need not be mentioned to the patient, and if mentioned should be explained as within normal limits. Only infrequently do symptoms arise from the words or action of a physician. It would appear to the writer to be as much of a crime to produce in a normal individual heart symptoms due to fear as it would be to miss a case of marginal organic heart disease.

The interpretation of the electrocardiogram showing only minor variation from normal is fraught with danger. While evidence of important abnormality should never be disregarded, minor variations such as slight ST depressions and PR prolongations and small Q waves should not be emphasized, especially when isolated findings, and had best not be mentioned to the patient unless serial determinations give additional significance.

The use of the photofluorogram reveals cases with contour change and as an isolated finding poses a problem of some importance which may get the diagnostician into deep water. Recently in case J. K. the isolated finding of an enlarged conus of unknown origin without proved left auricular or other enlargement was explained to the patient as being of no known clinical or health significance. It would seem that catheterization studies would seldom be indicated to explore a relatively minor finding except where certain congenital lesions are suspected. It is true that mitral or aortic or hypertensive configuration of the heart in early stages has at times disclosed disease not previously detected. Of the cardiac findings noted on a recent large chest x-ray survey, practically all cases coming to my personal attention were already on record.

The greatest skill of the practicing physician and cardiologist, in my opinion, is exercised when he sits down with the patient and takes time to listen to his complaints and story and to establish the relative importance of this or that factor, such as nervous and emotional tension and physical effort. For although most people are fairly uniform in perception of pain, they vary greatly in their tolerance of pain. No physician doubts that the severity of a pain is modified by the patient's interpretation of it. For in spite of limitations, from the history, together with the results of the physical examination, the leads are given that establish the presence or absence of heart disease. The patient who comes in complaining of breathlessness at rest, of inability to get a full breath, of sighing respiration, of heart consciousness or palpitation, of sticking or stabbing chest pains, all or some of which usually appear when quiet but not after effort, may be assured that his symptoms do not indicate heart disease. At the same time a careful physical examination, sometimes with ancillary procedures, is done. Often revealing factors come out on the first consultation, such as the illness or sudden death of a relative or friend from heart disease.

A series of cases with psychogenic arrhythmias has been reported by Fox and McKinlay,[1] and the significance of management was there discussed. The first case previously seen in the group of psychogenic arrhy-

thmias mentioned above was that of a student who developed auricular fibrillation at test times. As Fulton[2] has stated in discussing homeostasis, one can sit at a desk and overwork his heart just as much as if he were rowing a boat.

The multiple findings from the increased use of physical examinations, whether they reveal errors of habit, structural changes, or disturbance of organs or systems reflexly or otherwise, if soundly interpreted, will help to guard individual health and to stimulate interest in preventive medicine.

SUMMARY

1) The detection of latent heart disease through the examination of supposedly normal people and the evaluation of known disease in those actively employed raise questions about realistic methods of case discovery and about adequate interpretation of individual findings.

2) The individual who of his own choice seeks health advice from his physician is conditioned for the greatest benefit, granted that due diligence and interest are exercised by the examiner.

3) The history of previous illness, although difficult to obtain in group examinations, should be explored for further leads in case detection.

4) Certain physical findings such as the early diastolic murmur of aortic insufficiency or the pericardial friction rub may indicate the major abnormality.

5) The findings of mass screening methods such as routine chest x-ray films, electrocardiograms and other laboratory procedures should always have individual interpretation and must not in themselves be considered a complete diagnosis. Minor variations from normal have sometimes received unwarranted emphasis.

6) The manner in which special follow-up examinations are made and in which the information is conveyed to the individual may determine the development of undue fear of cardiac disease and of iatrogenic disturbance or the freedom therefrom.

7) The praise-worthy tendency in industry to use physically defective individuals, including cardiac cripples within the range of their capacity, demands that great care be given in estimating that capacity not only subjectively but objectively where possible.

8) Individualized attention to abnormal findings whether indicative of errors of habit, functional disturbance, or of organic disease will largely determine personal and public health value of group examinations.

RESUMEN

1) El descubrimiento de la enfermedad cardiaca latente por el examen de las personas aparentemente sanas y la evaluación de la enfermedad conocida en los empleados activamente, presenta problemas acerca de los métodos realistas para el descubrimiento de los casos y la adecuada interpretación de los hallazgos individuales.

2) El individuo por su propio criterio, busca el consejo médico, se bene-

ficia grandemente de la diligencia y el interés ejercidos por el que lo examina.

3) La historia de enfermedades anteriores, aunque difícil de obtener en los exámenes en grupos, debe explorarse para mayores guías en el descubrimiento de los casos.

4) Ciertos hallazgos físicos tales como un soplo al principio de la sístole propio de insuficiencia aórtica o el frotamiento pericárdico, pueden indicar una anormalidad mayor.

5) Los hallazgos de los métodos de investigación en masa, tales como las películas de tórax, electrocardiogramas y otros procedimientos de laboratorio, deben siempre sujetarse a interpretación individual y por sí solos no deben considerarse suficientes para el diagnóstico. Variantes menores de lo normal, algunas veces han merecido énfasis injustificado.

6) La manera como los exámenes de seguimiento de los casos se conducen cuando proporcionan al enfermo, pueden conducir a indebido teror de enfermedad cardiaca produciendo también alteración en la libertad de acción de fuente iatrogénica.

7) La tendencia laudable en la industria de usar personas físicamente defectuosas incluyendo cardiacos parcialmente incapacitados, necesita que se tenga gran cuidado para estimar la capacidad no sólo subjetiva, sino objetivamente cuando sea posible.

8) La atención individualizada a los hallazgos anormales ya sean indicadores de error en las costumbres, trastorno funcional o enfermedad orgánica, determinarán gruesamente el valor personal y público de la salud de los grupos examinados.

RESUME

1) La découverte d'affections cardiaques latentes lors de l'examen d'individus apparemment normaux et l'estimation de l'importance de la maladie, lorsqu'el le est connue, chez les gens qui ont une activité normale, posent certaines questions au sujet des méthodes pratiques d'examens systématiques, et sur la valeur réel le des constatations faites chez chaque individu.

2) Celui qui choisit son médecin de son propre gré se trouve dans les meilleures conditions puisque celui qui l'examine se doit de lui consacrer toute son activité et tout son intérêt.

3) Les antécédents du malade, bien que difficiles à connaitre lorsqu'il s'agit d'examens systématiques devraient être bien précisés pour prendre toutes les dispositions nécessaires au cas où une affection soit découverte.

4) Il est des constatations cliniques, comme le souffle diastolique précoce de l'insuffisance aortique ou le frottement péricardique qui traduisent l'existence d'une altération importante.

5) Les découvertes d'examens systématiques, tel les que la radiographie thoracique, l'électro-cardiogramme, et les autres recherches de laboratoires doivent être toujours interprétées d'une façon individuelle et ne doivent jamais être considérées comme suffisant à elles seules à faire le diagnostic. On à parfois attribué une valeur inconsidérée à des modifications minimes.

6) Les personnes qui ont subi un examen systématique peuvent être

saisies de la crainte d'être atteintes d'une affection cardiaque et aliéner ainsi leur liberté de comportement, si les examens sont mal conduits et les résultats maladroitement communiqués.

7) Dans l'industrie apparait maintenant la précieuse tendance de permettre une certaine activité aux individus physiquement déficients, y compris les cardiaques. Elle exige que soient estimées avec le plus grand soin les possibilités subjectives et quand c'est possible même objectives du malade.

8) En examinant des individus déterminés, on pourra constater des anomalies consécutives à des erreurs de régime, à des déficiences momentanées ou à des maladies organiques. Ces constatations seront d'un grand appoint pour juger l'importance qu'ont les examens systématiques, tant pour l'individu que pour l'hygiène sociale.

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Changing Trends in the Treatment of Lung Abscess*

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Introduction

The advent of antibiotics, notably penicillin, has admittedly had a profoundly favorable effect on the prognosis of lung abscess. Although few figures are available, the opinion is repeatedly expressed in the literature and by chest specialists in general that the incidence of lung abscess is decreasing. Our own figures show a numerical increase but a proportional decrease in comparison to patients seen in other categories. While this decrease is due largely to the prompt and effective use of antibiotics in aborting the pneumonic infections that frequently went on to lung abscess in the past, due credit must be given to the wider acceptance of early bronchoscopy as a therapeutic measure. Certainly the morbidity of the average case is decidedly reduced by antibiotics. The extremely ill, highly toxic and febrile patient so frequently seen formerly is now rarely encountered. The antibiotics in such cases not only afford symptomatic palliation for the patient, but also buy valuable time for the surgeon in which to effectively apply therapy.

Increasingly commonly, however, this palliative effect is obscuring diagnosis, especially of underlying primary pathology such as carcinoma, and is delaying necessary surgical intervention. As a result even more chronic abscesses are being encountered today than before chemotherapy was available, and more resection surgery is necessary. Fortunately the tremendous impetus the antibiotics have given pulmonary surgery makes possible the successful salvage of most of these cases, although unfortunately at the expense of lung tissue.

When Allen and Blackman[1] presented their collected figures in 1936, the gross mortality of the 2,114 cases was 34.3 per cent. The medical mortality was 34.4 per cent and the surgical 34.2 per cent. As late as 1948 Smith[2] collected 2,166 cases from the literature from 1936 to 1946, and showed a gross mortality only one-tenth of 1 per cent lower than that of the previous report. The medical and surgical mortality were not significantly reduced, as is shown in Table I. The high percentage of chronicity is also of great significance, and serves to emphasize the seriousness of the disease.

TABLE I: MORTALITY FIGURES

Author	No. Cases	Gross Per cent	Medical Per cent	Surgical Per cent
Allen and Blackman (to 1936)	2,114	34.3	34.4	34.2
Smith (1936-1946)	2,166	34.2	34.7	32.7

*Presented at the Second International Congress on Diseases of the Chest, American College of Chest Physicians, Rio de Janeiro, Brazil, August 24-30, 1952.

This gloomy picture has materially brightened, especially in the therapy of selected groups of patients. To Neuhof, Tourhoff and their associates[3-8] must go the credit for demonstrating to the profession the desirability of early drainage of the acute putrid abscess. Their mortality in 165 cases was only 2.43 per cent, and their chronicity rate only 13.4 per cent by strict criteria on a long term basis. Following this signal advance, the reports of Lindskog,[9] Sweet,[10] Glover and Clagett,[11] Shaw and Paulson,[12] Kent and Ashburn,[13] Klepser and Davis,[14] and more recently Bosher[15] reflect decided improvement in surgical therapy. Shaw and Paulson's report of 52 resections for chronic putrid lung abscess with but two deaths, a mortality of 3.8 per cent, is particularly impressive. Weisel, Raine and Owen[16] have contributed an excellent report on the combined medical and surgical treatment of the acute putrid lung abscess, and Stilvelman and Kavee[17] an optimistic picture of the treatment of acute putrid lung abscess with penicillin and sulfadiazine. Numerous other articles attest the trend.

Criteria of Report

It was thought that a series of cases consecutively seen and treated both medically and surgically by the same individuals would be of interest, and lend further weight to these excellent reports. A total of 401 abscesses came under our care from 1941 to 1952. Bronchiectasis, tuberculosis, and mycotic infections were excluded, but cases of suppurative bronchopneumonia included, as in Smith's figures. Listed in the overall tabulation are six cases of embolic abscess with pyemia, not a comparable group as to abscess therapy. Cases of pulmonary neoplasm with definite abscesses or secondary suppurative pneumonia are included in order to emphasize the importance of malignancy as a causative factor in the disease. The remaining 218 cases of pulmonary abscesses and suppurative pneumonia constitute the basis for this report. The 401 cases are presented in Table II.

TABLE II: TYPE OF CASE

Lesion	Number	Per cent
Lung abscess or suppurative pneumonia	218	54.4
Carcinoma with secondary suppuration	174	43.4
Adenoma with secondary suppuration	3	0.7
Embolic abscess with pyemia	6	1.5
TOTALS	401	100.0

The incidence of carcinoma as an etiological agent in our series rose from 11 per cent in 1941 to 52 per cent by 1948. This remarkably high occurrence of carcinoma makes it imperative that every case of lung abscess be thoroughly investigated for malignancy.

The 218 cases used in this report were classified as putrid or nonputrid, and as lung abscesses or as suppurative pneumonias, as shown in Table III.

These last differentiations are of course at times rather difficult. As

TABLE III: PROPORTION OF CASES

Type of Suppuration	Number	Per cent	Totals	Per cent
Putrid lung abscess	125	82.8	151	69.2
Putrid suppurative pneumonia	26	17.2		
TOTALS	151	100.0		
Nonputrid lung abscess	24	35.8	67	30.8
Nonputrid suppurative pneumonia	43	64.2		
TOTALS	67	100.0	218	100.0

Smith has pointed out, the sputum has no odor in a putrid abscess during its early development. Under the influence of antimicrobial agents a putrid abscess may become nonputrid rather rapidly, or the putrid odor never become detectable when therapy is started early. We have also seen bronchoscopy change a putrid abscess to a nonputrid one. Smith has repeatedly pointed out that bacteriological studies are the important diagnostic criteria in determining the type of abscess. In addition, the 218 cases were listed as acute up to six weeks, subacute from six weeks to six months, and as chronic when the duration was over six months, as suggested by Shaw.[18] This is purely an arbitrary division, and has obvious disadvantages. The detailed listing is not given in this report, but was used for study in analyzing the results for "Discussion."

In order to show the influence of antibiotics on both the medical and surgical treatment of abscesses, the group was divided into a preantibiotic and a postantibiotic category, using July, 1946, as an arbitrary dividing point. This was the approximate time at which penicillin came to be used in adequate dosage in our cases. During the preantibiotic period 90 cases were treated, and during the postantibiotic 135, making the groups relatively comparable numerically. No attempt was made to eliminate cases who refused surgery, as these serve as controls to further evaluate medical treatment. Inadequately treated cases were included so that the series would reflect the actual situation encountered. In this respect, it is open to question as not reflecting the true potentialities of therapy. The figures on percentages of these two periods are shown in Table IV.

TABLE IV: CASES TREATED

Period	Number	Per cent
Preantibiotic (1941-1946)	90	40
Postantibiotic (1946-1952)	135*	60

*Seven preantibiotic cases retreated in postantibiotic period.

Of the 218 cases, 169 or 77.5 per cent were males, and 49 or 22.5 per cent females. Approximately 60 per cent had severe dental sepsis, a factor which we felt was exceedingly important etiologically. Of the abscesses involved

64.7 per cent were on the right side, with only 35.3 per cent involving the left. The right lower lobe was the most common site, being involved in 62 of the 218 cases. The susceptibility of this lobe to aspiration infection is of course well known. The next most common site was the right middle lobe, 48 cases being involved. This is felt to be explained by the frequency of block of the lobe as in right middle lobe syndrome.

Conservative Therapy

Much confusion has resulted in the literature from presenting medical and surgical therapy as competing treatments, whereas they are actually complimentary. Since the revolutionary concepts of Neuhof and Tourhoff were presented, the opinion has been expressed repeatedly by such authors as Glover and Clagett, and Klepser and Davis, that lung abscess is a surgical disease from the outset. We are in complete agreement, and feel that the poor results of medical treatment in the past are in no small part due to the failure to recognize this concept. We have been fortunate in most of the cases in our series in being able to apply either conservative or surgical therapy as we saw fit, although we have had the distressing experience shared by many other surgical contributors of seeing many of these patients far too late in the course of the disease after prolonged and inadequate antibiotic treatment. While our figures show half of the cases clearing under present day conservative management, we feel that statements contending that the antibiotics have made lung abscess almost entirely a medical problem are both fallacious and dangerous.

TABLE V: RESULTS — CONSERVATIVE THERAPY

Period	ABSCESS RESULTS		— — PATIENT STATUS — —		
	Good	Poor	Well	Chronic	Dead
Preantibiotic (60 cases)	38	22	32	14	14
Per cent	63.3	36.7	53.4	23.3	23.3
Postantibiotic (74 cases)	57	17	46	25	3
Per cent	77.0	23.0	62.2	33.8	4.0
TOTALS (134 cases)	95	39	78	39	17
Per cent	70.9	29.1	58.2	29.1	12.7

The results of conservative therapy in the pre- and the postantibiotic periods, with the totals for the group, are shown in Table V. Of particular significance is the marked reduction in mortality from 23.3 per cent in the preantibiotic era to 4.0 per cent in the postantibiotic period, and the corresponding improvement in good results obtained. In the acute and subacute groups the results have been strikingly better, both putrid and nonputrid abscesses responding favorably almost in direct proportion to the age of the abscess.

In spite of the decided improvement in results in the conservative group since the introduction of the antibiotics, there has been no reduction in the proportion of patients coming to operation. It must be remembered

that the group listed under conservative therapy, in most instances, are those who responded to medical treatment. The surgical cases are those who failed to respond.

Bronchoscopy is of prime importance, not only in the conservative group, but in all abscesses. We feel it is the initial step in the regimen, both for diagnostic and therapeutic reasons. Our 401 cases were bronchoscoped with only six exceptions. The high incidence of neoplasm in the group would seem to make the procedure imperative; however, segmental localization of the abscess, evaluation of the bronchial reaction, examination for foreign body, and procurement of bronchial secretions for bacteriological study in themselves justify the procedure. Therapeutically its early use can frequently abort a lung abscess in the formative stage or markedly improve the more advanced abscess by promoting bronchial drainage. In 32.1 per cent of the abscess series bronchoscopy was performed more than once, 382 procedures being carried out on the 218 patients.

In the preantibiotic period all patients were treated by bed rest and supportive treatment, and usually given sulfadiazine. Following the initial bronchoscopy all patients were placed on postural drainage and expectorants. Postero-anterior and lateral x-ray films were made at least at weekly intervals and surgery considered if improvement was not prompt and steady following the first bronchoscopy. Bronchoscopy was repeated if the patient again became febrile or failed to raise sputum adequately. All sputum was collected in the routine manner using glass containers, and the amount recorded daily. Arsenicals and iodides were occasionally added to the treatment in a small percentage of cases.

In the postantibiotic period since mid-1946, large doses of penicil usually 100,000 to 300,000 units every three hours, have been added to regimen. It has been our feeling that penicillin-G potassium given at frequent intervals has been more effective than the repository preparations. We frequently, however, administer procaine penicillin at bedtime so that the patient may be given uninterrupted rest. Aerosol penicillin is used routinely following the initial bronchoscopy, from 25,000 to 50,000 units being given every three hours during the day by means of an oxygen pressure-nebulizer. A shrinking agent, usually neo-synephrine or prothricin, is added to the nebulization. We have been particularly enthusiastic about the use of aerosol in all forms of bronchial infection, having had an experience in over 1,600 cases. In the abscess group the improvement has been particularly striking. We have seen the severe angry endobronchial reaction, so typical of pulmonary abscess, disappear within a period of three days with the use of inhalation therapy. At the present time we are investigating the use of terramycin by the aerosol route, but have had too limited an experience to express an opinion, except that the initial results are encouraging. As to the use of oral antibiotics, aureomycin in 500 milligram doses has been used in a small number of cases with good results. We have hesitated to use chloramycetin over prolonged periods, but have used it restrictedly with success. None of the oral preparations, however, have been as effective as penicillin by the aerosol and intermuscular routes.

Streptomycin, however, has been of material help in indicated cases, such as Friedlander's pneumonias.

One other point deserves special mention, namely, the effective treatment of the dental sepsis found in so many of these patients. This should be done as soon as feasible after the patient enters the hospital, and certainly before resection therapy.

Open Drainage Therapy

Table VI tabulates the results obtained in the preantibiotic and the postantibiotic era.

TABLE VI: RESULTS — DRAINAGE THERAPY

Period	ABSCESS RESULTS		— — PATIENT STATUS — —		
	Good	Poor	Well	Chronic	Dead
Preantibiotic (16 cases)	10	6	10	4	2
Per cent	62.5	37.5	62.5	25.0	12.5
Postantibiotic (20 cases)	15	5	13	4	3
Per cent	75.0	25.0	72.0	16.0	12.0
TOTALS (36 cases)	25	11	23	8	5
Per cent	69.4	30.6	63.9	22.2	13.9

Here again good results were in direct proportion to the age of the abscess. There was no mortality in the acute group drained, and only one case later showing a residual defect demanding lobectomy. In the subacute abscesses there was one poor result, the others being in the chronic group, all of whom were drained early in the preantibiotic era before resection became firmly established as the definitive therapy.

There has been a decided trend away from drainage in lung abscess in all series recently reported. The procedure is now reserved in our hands for the fulminating rapidly progressive abscess in the poor risk patient, or the uncomplicated isolated peripherally located acute abscess where a good result can be anticipated. Nevertheless, it must be remembered that the results from open drainage have been excellent when correctly applied. The overall results in the postantibiotic era are still only 75 per cent good, however, and 16 per cent remain ill.

Tourhoff et al have provided ample evidence that unsuspected pulmonary defects can remain silent for long periods following drainage. We agree that it is imperative that all cases have a bronchogram at a later date to determine if such residual foci remain.

In both the preantibiotic and the postantibiotic periods a one-stage procedure has been used, only seven of our cases requiring a two-stage procedure. It has been our practice for some years to suture the visceral pleura to the parietal if pleural symphysis is not present. The abscess is then opened in the routine manner. In no case has an empyema resulted, even before the use of antibiotics. Admittedly there are relatively few of these cases, as most abscesses are peripheral and have a cone of adhesions. We

have, however, encountered an appreciable number of abscesses closely approximating the pulmonary or lobar hilus. These of course are rarely suitable for drainage.

Accurate localization of the abscess on postero-anterior and lateral six foot films is of course a prerequisite. Usually only one segment of rib is removed in the drainage procedure, this being over the center rather than the dependant portion of the abscess. It is felt that abscesses heal concentrically, and that a tube so placed will be optimal as the abscess reduces in size. In the preantibiotic era, zinc peroxide powder or Melaney's paste, was used routinely on the packing, but with the antibiotics we have rarely found this necessary, and have used washed iodoform gauze. The packing is used until all slough is removed and the abscess begins to reduce in size satisfactorily. The tube is not taken out until the cavity is completely obliterated, as in empyema.

Needless to say, cultures are taken of each abscess, and the antibiotics selected accordingly. If penicillin is the drug of choice, usually the repository preparations suffice, as prompt reduction in temperature and toxicity follow the drainage as a rule. Antibiotics are discontinued when the clinical and x-ray signs show satisfactory progress.

Resection Therapy

The results of extirpation therapy are given under Table VII.

TABLE VII: RESULTS — RESECTION THERAPY

Period	ABSCESS RESULTS Good	Poor	— — PATIENT STATUS — — Well	Chronic	Dead
Preantibiotic (14 cases)	10	4	8	2	4
Per cent	71.4	28.6	57.1	14.3	28.6
Postantibiotic (41 cases)	40	1	39	1	1
Per cent	97.6	2.4	95.2	2.4	2.4
TOTALS (55 cases)	50	5	47	3	5
Per cent	90.9	9.1	85.4	5.5	9.1

Most striking is the reduction in mortality in the postantibiotic group, only one case being lost, giving a mortality percentage of 2.4 per cent. Only one case is chronic with a small residual empyema space. The combined figures are by far the best of any type of therapy.

There were 41 lobectomies, five pneumonectomies, and nine segmental lobectomies (Table VIII).

Our indications for resection coincide with those of Glover and Clagett. As have others, we have been well aware of the increasing number of chronic lung abscesses resulting from over-enthusiastic dependence on antibiotics. Rosenfeld and Holcomb,[19] in an unpublished report, found that 84 per cent of the 55 cases seen at Vanderbilt University from 1945 to 1950 were chronic, a striking increase over the previous two five-year periods.

All patients have been prepared for surgery with two days of intensive

TABLE VIII: COMPLICATIONS AND MORTALITY OF RESECTION

Procedure	Cases	Empyemas	B-P Fistulae	Deaths
Pneumonectomy	6	5	1	2
Per cent	10.9	50.0	16.6	33.3
Lobectomy	40	6	5	3
Per cent	72.7	15.0	12.5	7.5
Segmental Lobectomy	9	0	0	0
Per cent	16.4	0.0	0.0	0.0
TOTALS	55	9	6	5
Per cent	100.0	16.4	10.9	9.1

penicillin therapy intermuscularly usually accompanied by streptomycin. Aerosol antibiotics have been used in all cases since early 1947. We feel they definitely enhance the safety factor and reduce the hazard of post-operative complications. Preoperative transfusions are usually necessary, as these patients are notoriously poor operative risks, with secondary anemia and cachexia.

Individual ligation technique was used in the entire series with the exception of one case, where a wedge segmental resection was necessary in an anterior segment of a left upper lobe in order to avoid sacrificing the entire lung because of hilar involvement. We heartily subscribe to the dictum of the desirability of conserving lung tissue, and make every effort to remove the smallest amount possible. Frequently this considerably increases the technical difficulties of operation. We have, however, been able to apply segmental resection more and more frequently.

In this respect we are particularly enthusiastic about the simple excision technique outlined by Holman.[20] We have found it possible to peel a chronic abscess out of surrounding good lung tissue with very little bleeding, as the wall of the abscess provides a cleavage plane that permits relatively easy dissection. Recently we have had the experience of removing such an abscess from the hilus, only sacrificing a small portion of the lower lobe, where the abscess lay, and the middle lobe, which was blocked. By usual operative standards, pneumonectomy would have been necessary, a procedure this 52 year old seriously ill female would have been probably unable to tolerate. We have been using a similar technique in tuberculous resection for some time, but have only applied it to lung abscess since Holman's contribution. While we believe it will probably increase the incidence of complications, particularly empyema and bronchopleural fistula, we feel the preservation of lung tissue resulting warrants the risk.

As indicated in Table VIII, the complication of empyema remains a serious problem. We have had an incidence of 18.2 per cent although five of the nine cases occurred in the preantibiotic era. Three of the five pneumonectomies developed empyema. In one of these cases, intrapleural installation of penicillin affected a cure, in spite of the fact that thick pus had formed. No bronchopleural fistula was present, and no thoracoplasty has been done. The patient has continued active work, and reports an

increasing work tolerance over the ensuing five years. In the other two cases of pneumonectomy developing empyema, open drainage and thoracoplasty were necessary. In only one of the three cases was bronchopleural fistula present.

Of the six cases of lobectomy developing empyema, four had bronchopleural fistulae requiring drainage. In one case of left upper lobectomy with a residual space, a small stump abscess eroded six weeks postoperatively directly into the overlying pulmonary artery, with exsanguinating hemorrhage. Since that time it has been our practice to interpose a barrier of free pleura tied in a roll over the stump with the bronchial sutures, as shown in Figure 1.

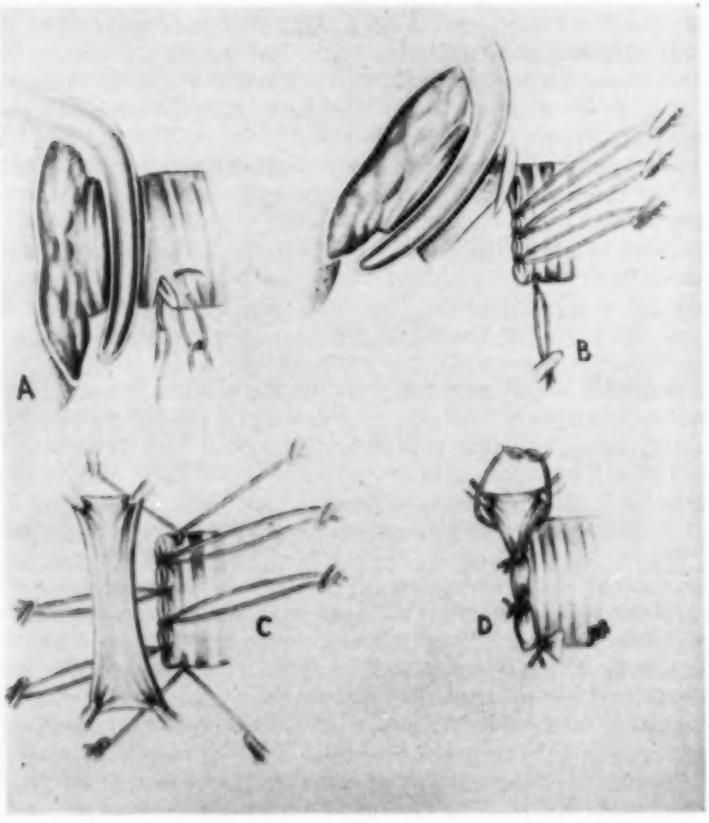

FIGURE 1: *Technique of Bronchial Closure.*

A, Division of bronchus proximal to clamp.
B, Insertion of bronchial sutures as division proceeds.
C, Free pleural graft held over closed bronchus.
D, Graft tied in place over stump with bronchial sutures.

This technique has apparently been the factor that has reduced the incidence of bronchopleural fistulae in our entire resection series. We feel that it is particularly important in lung abscess.

Although the series of segmental resections is small, no empyemas occurred. Certainly the factor of early postoperative space obliteration is important. Closed drainage tubes with active suction should be reinserted promptly if space persists.

Discussion

For statistical purposes, in order that the figures would be comparable with other published reports, the surgical therapy was combined in Table IX.

TABLE IX: RESULTS — SURGICAL THERAPY
(Drainage and Resection)

Period	ABSCESS RESULTS Good	Poor	— — PATIENT STATUS — — Well	Chronic	Dead
Preantibiotic (30 cases)	20	10	18	6	6
Per cent	66.7	33.3	60.0	20.0	20.0
Postantibiotic (61 cases)	55	6	52	5	4
Per cent	90.2	9.8	85.2	8.2	6.6
TOTALS (91 cases)	75	16	70	11	10
Per cent	82.4	17.6	76.9	12.1	11.0

In comparison to conservative therapy, there is a significant reduction in the poor results and in the chronicity figures, especially in the post-antibiotic era where the poor results dropped from 23.0 to 9.8 per cent, and chronicity from 33.8 to 8.2 per cent.

The decided improvement in surgical figures in the postantibiotic period is of course due to three factors in addition to the use of antibiotics: improved surgical techniques, improved blood replacement therapy, and improved anesthesia. While the best results with conservative therapy are obtained in the earliest cases, the majority of the resection cases are in the chronic group, as in other series, with only a small number of acute and a slightly larger number of subacute cases requiring extirpation.

It would seem that if generalizations can be made from the series, that acute putrid abscess can now be successfully treated conservatively in most cases, with an occasional case requiring open drainage or resection. Subacute putrid abscess often responds well to conservative treatment, occasionally is suitable for open drainage, and fairly often requires resection. Chronic lung abscess rarely does well with conservative therapy or with drainage, but shows excellent results with resection.

Acute putrid suppurative pneumonias also do well with conservative treatment and of course are not suitable for drainage. Resection has not been necessary in our series but conceivably might be. In the subacute putrid pneumonias, however, an occasional resection is necessary. We have had good results with conservative treatment here as well, and of course

have never resorted to drainage. When suppurative pneumonia is chronic, resection is the only treatment of choice and gives admirable results.

Acute nonputrid lung abscess usually responds well to conservative treatment. We have had good results, however, with drainage therapy in a limited number of cases, contrary to the experience of Tourhoff and Neuhof, and of Smith. When this type of abscess is subacute, it still responds well to conservative treatment, and not too badly to drainage. Resection has not been necessary in our series, but well might be. When these abscesses are chronic, resection is of course the best treatment as in the putrid group. We have occasionally drained one successfully however. Results with conservative treatment are poor.

The best treatment in acute nonputrid pneumonias is a conservative regimen with drainage never suitable and resection rarely necessary. In the subacute group of these, good results can also be obtained with conservative treatment but the occasional case will be found needing resection. Again in the chronic nonputrid pneumonia group, resection is the only treatment worthy of consideration.

It should be reiterated that lung abscess should receive surgical consideration from the outset. The physician should be alert to the desirability of early bronchoscopy in all pneumonias failing to promptly clear, and aware of the necessity of prompt surgical intervention if the conservatively treated abscess does not steadily and rapidly improve.

SUMMARY

1) Antibiotics have improved the prognosis, and have reduced the incidence and morbidity of lung abscess, but have by no means eradicated the disease. They have often delayed diagnosis and treatment.

2) Lung abscess is a surgical problem from the outset, and demands close cooperation between the internist and the surgeon.

3) Carcinoma as an etiological factor in lung abscess is increasingly common, and should invariably be considered in the diagnosis.

4) The use of antibiotics has greatly increased the efficacy of conservative therapy in the early abscess.

5) Bronchoscopy should be performed in every case of pulmonary abscess, both for diagnosis and as an integral part of conservative treatment.

6) Drainage therapy, once the surgical treatment of choice, is now largely reserved for the progressive acute abscess in the poor risk patient, and in the good risk patient where a good result can be accurately anticipated. Antibiotics have, however, improved the overall good results in drainage, and the procedure should by no means be discarded.

7) Resection therapy is the definitive surgical procedure in most abscesses, particularly since antimicrobial agents have come into use. Mortality is gratifyingly low, and results excellent. It would seem to be the answer to the problem of the chronic abscess.

8) When resection therapy is used, segmental resection or lobectomy are preferable to pneumonectomy, if at all feasible, because of the lower mortality and complications, and in order to preserve maximum function.

9) Simple excision as a technique of segmental resection in chronic lung abscess appears at this time to offer great hope for effective conservation of lung tissue.

10) Although surgical therapy produces better results than conservative, the regimens are complimentary rather than competitive.

11) Intermuscular, oral, and aerosol antibiotics, notably penicillin, have materially improved the prognosis in resection. The incidence of empyema, however, is far too high, and should be improved.

RESUMEN

1) Los antibióticos han mejorado el pronóstico y disminuido la frecuencia del absceso pulmonar pero de ninguna manera han hecho desaparecer la enfermedad. A menudo a ellos se debe un retardo en el diagnóstico y en el tratamiento.

2) El absceso pulmonar es un problema quirúrgico desde un principio y requiere estrecha cooperación entre el internista y el cirujano.

3) El carcinoma como un factor etiológico en el absceso del pulmón es cada vez mas frecuente y debe considerarse invariablemente en el diagnóstico.

4) El uso de los antibióticos ha aumentado la eficacia del tratamiento conservador al principio del absceso.

5) En todo caso debe hacerse la broncoscopia en el absceso pulmonar tanto para el diagnóstico como formando parte del tratamiento integral conservador.

6) La canalización quirúrgica que alguna vez fué el tratamiento de elección se reserva ahora mas bien para el absceso agudo progresivo en los enfermos en malas condiciones y en los de mejor estado cuando un buen resultado puede seguramente preverse. Los antibióticos sin embargo han mejorado en general los resultados de la canalización y de ninguna manera hay que descartar el procedimiento.

7) La resección es el procedimiento quirurgico definitivo en la mayoría de los abscesos especialmente desde que se cuenta con los agentes terapéuticos antimicrobianos. La mortalidad es satisfactoriamente baja y los resultados excelentes. Parece que es la solución al problema del absceso crónico.

8) Cuando la resección se usa, la segmentaria o la lobectomía son preferibles a la neumonectomía si es posible, a causa de la mortalidad menor y las escasas complicaciones y también para conservar el máximo de la función.

9) La excisión simple como una técnica de la resección segmentaria en el absceso pulmonar crónico parece ahora ofrecer mayor esperanza en lo referente a la conservación efectiva de tejido pulmonar.

10) Aunque la terapéutica quirúrgica produce mejores resultados que la conservadora ambos metodos se complementan mas no compiten.

11) Los antibioticos intramusculares, orales, en aerosol, y especialmente la penicilina han mejorado materialmente el pronóstico de la resección. La frecuencia del empiema, sin embargo, sigue demasiado alta y debe mejorar.

RESUME

1) Les antibiotiques ont amélioré le pronostic et réduit la fréquence et la morbidité des abcès pulmonaires, mais n'ont en aucune façon fait disparaitre l'affection. Ils ont souvent retardé le diagnostic et le traitement.

2) Dès le départ, l'abcès pulmonaire est un problème chirurgical qui demande une étroite coopération entre médecin et chirurgien.

3) La fréquence du cancer en tant que facteur étiologique dans l'abcès pulmonaire s'accroit constamment. Se diagnostic doit toujours être pris en considération.

4) L'emploi des antibiotiques a grandement augmenté l'efficacité du traitement médical simple en cas d'abcès précoces.

5) La bronchoscopie devrait être pratiquée dans chaque cas d'abcès pulmonaire, à la fois comme élément de diagnostic et comme élément thérapeutique.

6) Le simple drainage de l'abcès est maintenant réservé aux abcès aigus évolutifs, pour les malades qui semblent peu opérables. Il peut être également employé chez les malades pour lesquels le pronostic est assez grave, quand on peut prévoir qu'on en obtiendra de bons résultats. Toutefois, les antibiotiques ont notablement augmenté les bons résultats du drainage et en aucun cas, ce procédé ne doit être abandonné.

7) La résection est le procédé chirurgical parfait dans la plupart des abcès, particulièrement depuis qu'on utilise les agents antimicrobiens. La mortalité est très abaissée et les résultats excellents. Elle semblerait être la réponse au problème des abcès chroniques.

8) Quand on utilise la chirurgie d'exérèse, il faut préférer la résection segmentaire ou la lobectomie à la pneumonectomie. Si ces opérations plus limitées sont possibles, elles donnent une mortalité plus faible, moins de complications et préservent au maximum la fonction pulmonaire.

9) La simple excision qui peut être utilisée comme technique de résection segmentaire pour les abcès chroniques du poumon peut sembler offrir un grand espoir de conservation importante du tissu pulmonaire.

10) Bien que la thérapeutique chirurgicale donne de meilleurs résultats que le simple traitement conservateur, les deux moyens semblent plutôt devoir se compléter que se contrarier.

11) Les antibiotiques administrés par voir intramusculaire par voie bucale ou en aérosols, particulièrement la pénicilline, ont amélioré le pronostic des résections. La fréquence des pleurésies purulentes cependant est trop élevée et devrait être améliorée.

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The Usefulness of the Hotchkiss-McManus Stain for the Diagnosis of the Deep Mycoses

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The isolation of pathogenic fungi is more difficult than most physicians appreciate and the average hospital bacteriology laboratory approaches the problem in a half-hearted and uninstructed manner. Too often the diagnosis is a "coup de grâce" administered by the pathologist at the clinical pathological conference.

As a matter of fact, a considerable number of deep mycoses must go unrecognized each year despite the provision of adequate material to the pathologist for at least a tentative diagnosis. There are a number of reasons for this state of affairs; the two which are outstanding implicate both the clinician and the pathologist.

Many physicians develop tubular vision when confronted with patients who present the problems of chronic lymphadenopathy, "cold" abscesses, or persistent pulmonary infiltration. Requisitions for laboratory studies on such patients are likely to bear the provisional diagnosis of tuberculosis and to request a search for acid-fast bacilli or evidence of neoplasia. If a series of negative reports is received from the laboratory, the patient may be treated for tuberculosis on clinical grounds or perhaps discharged. All too often the clinician requests cultural studies for fungi as a last resort and somewhat reluctantly.

On the other hand, the pathologists have been equally hampered by tradition. The student is still admonished to use the low power objective of his microscope and a high power cerebral cortex. We realize that pathologists are busy people and particularly those in large hospitals where a great many routine biopsy sections must be read during a day. Most histopathological diagnosis is done, and adequately, with the low and medium power objectives. When such a microscopic examination seems quite compatible with the clinical diagnosis which accompanied the specimen, it is not surprising that both the pathologist and the clinician should be satisfied with the report "chronic granuloma compatible with tuberculosis."

Unfortunately, so much that seems routine is not, and one may be trapped by the obvious. The cases which we present are all examples of pulmonary mycoses which were initially undiagnosed or misdiagnosed. It is our purpose to bring to general attention a particular procedure which greatly reduces the amount of time required for adequate study of a biopsy

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and which reveals fungi in embedded tissues with a clarity not previously obtainable.

The periodic acid-Schiff method is now six years old and similar techniques which are less precise for fungi antedate it by about 15 years. Nevertheless, it is only within the last 18 months that the method has appeared in journals primarily read by clinicians. Some within our acquaintance have tried the method and rejected it because of poor results. We have used this method for over a year in a routine manner. We have not found it to be capricious, but agree that it requires a basic knowledge of microtechnique which any laboratory technician who is adequately trained and sufficiently interested can carry out without particular attention to individual care of each slide.

The Schiff reagent is prepared by decolorizing basic fuchsin with hydrochloric acid and anhydrous metabisulfite. This forms a colorless fuchsin sulfurous acid which will combine with aldehydes. When this combination takes place, the reagent is recolorized by the addition aldehyde and if the aldehyde is relatively insoluble, the substance containing it will be stained varying shades of pink-to red-to purple.

Hotchkiss and McManus, working independently, found that periodic acid could be used to oxidize certain carbohydrates, mucoproteins, and glycoproteins to aldehydes which could then be colored by the Schiff reagent. The pathogenic fungi and some bacteria contain material which, after oxidation with periodic acid, will form colored addition compounds with the Schiff reagent.

In order to be as objective as possible and to illustrate without exaggeration the advantages of the stain in the presented cases, we have selected a method which is not open to the criticisms of either chance or selection. With the exception of our first case, slides were prepared from serial sections and a single section stained by the periodic acid-Schiff method. After the area of involvement was located in this lide, the succeeding slide was stained by a routine hematoxylin-eosin method. The area of involvement was then found on this slide and photographed by appropriate powers of magnification. This area was localized on the slide and the cover slip removed for restaining by the periodic acid-Schiff method. The same area on the same slide was photographed after the application of this stain. This approach obviates the possible criticism, when photographing successive sections, that the area photographed may have contained abundant organisms in one section and not in the other. This is particularly true when dealing with such small organisms as Histoplasma capsulatum. Illustrations are of the same sections and whatever difference exists in obviousness of the etiology is due entirely to the method of staining. It should be remembered that the black and white illustrations cannot adequately reproduce the color contrast between the red organisms and the green background which is so large an advantage of this method.

The circumstances surrounding case one made it impossible for us to follow the procedure just outlined. This patient was the first on whom the method was tried and it was not until six months later that we decided

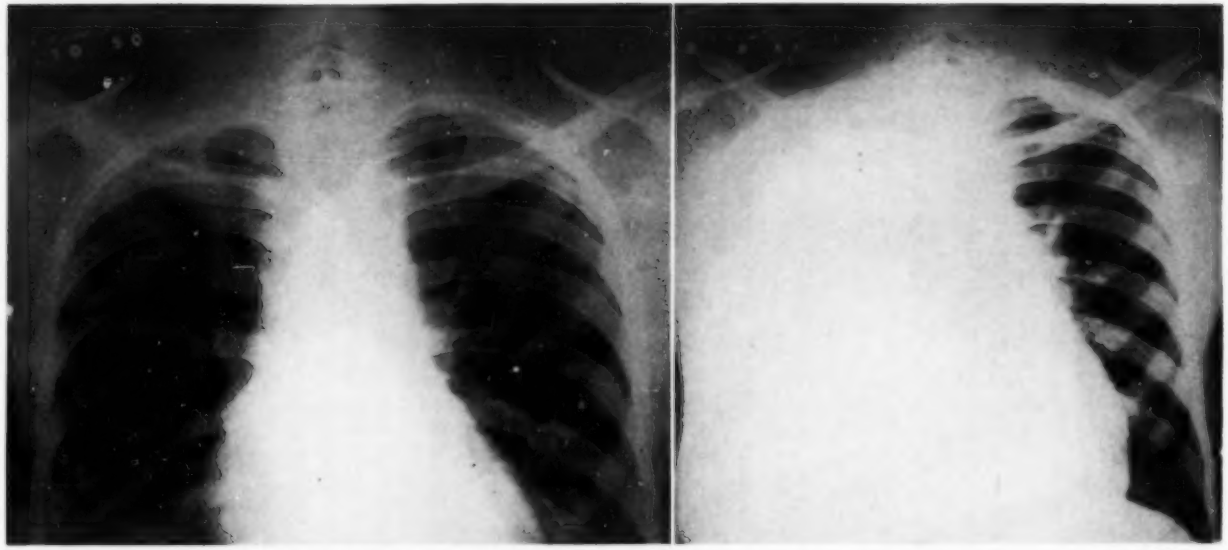

FIGURE 1A FIGURE 1B

Figure 1: Pulmonary Moniliasis. (1A) This x-ray film, taken on admission, reveals widening of the supracardiac shadow due to mediastinal lymphadenopathy. (1B) Right pleural effusion was present six weeks before this film was taken. Fluid production was rapid and had to be removed frequently.

to write this article. The finding of enormous numbers of Candida in what was believed to be a piece of bronchus would not have been given so much significance had we not found areas of moniliasis in two of the lymph node biopsies. We were, as a matter of fact, recutting these biopsies serially in a search for Blastomyces and all sections were stained with periodic acid-Schiff reagent.

When the small areas of moniliasis were found, the paraffin block of the expectorated tissue, thought to be bronchus, was sought. It could not be found, and after a careful review of the original section, it was restained with periodic acid-Schiff reagent without first being photographed. To complete the comedy of errors surrounding this case, six months elapsed before it was felt that this paper would be desirable and photography undertaken. To our chagrin, the periodic acid-Schiff stained node sections had faded completely and our efforts at restaining were unsuccessful. The other lymph node biopsies were negative for fungi.

Figure 1A is the chest x-ray film of a 22 year old negro who was admitted to the Charity Hospital at New Orleans in December of 1950. He was admitted with the chief complaints of painful lumps in both supraclavicular regions, pain in the left anterior chest on deep breathing, and paroxysmal cough. He was a saw mill worker who dated his illness to the onset of a painful tumor in the right infraclavicular region which he described as being like a small egg. After taking sul-

FIGURE 2. Pulmonary Moniliasis. Typical pseudomycelium and blastospores may be seen in this high power field taken in an area of fibrous connective tissue and smooth muscle adjacent to a medium-sized vein.

fonamides prescribed by his physician, the swelling subsided moderately and his general sense of well-being improved. Shortly after return to work he developed subscapular pain which was aggravated by movement, together with cervical lymphadenopathy, fever, anorexia, and general malaise. Sulfonamide therapy lessened his discomfort but he soon developed exertional dyspnea and anterior chest pain.

A low grade fever was present upon admission to the hospital but subsided within two days. With the exception of slight temperature elevations following biopsy procedures, he was afebrile until the fifth week of hospitalization. Thereafter a low grade fever was present on at least several days of each week.

The initial laboratory studies revealed moderate anemia with rapid sedimentation rate, normal total and differential white blood cell count, and Mantoux test which was negative at a 1:1000 dilution but positive at 1:100. Cervical node biopsy was negative on smear and culture for acid-fast bacilli and was reported histopathologically as "chronic nonspecific lymphadenitis." No significant abnormalities were found in the sternal marrow.

Two weeks after admission there was considerable increase in the size of the lymph nodes and several firm and tender nodes were palpated in the left axilla and beneath the left pectoralis major. The enlargement of the matted cervical nodes produced the picture of so-called "bull neck." The spleen was not palpable. Wheezing rhonchi became audible. Three weeks after admission an axillary lymph node was biopsied and reported as showing chronic lymphadenitis. Gastrointestinal x-rays, sigmoidoscopy, and bone survey by x-ray were all negative. The electrocardiogram was not outside normal limits. Since it was the opinion of the attending staff that tuberculosis was the probable diagnosis, he was treated with streptomycin and para-aminosalicylic acid.

A left supraclavicular lymph node biopsy carried out four weeks after admission added no new information. At this time the skin tests were repeated: the standard histoplasmin was 2 plus, the blastomyces vaccine 1:1000 and the coccidioidin 1:100 were negative.

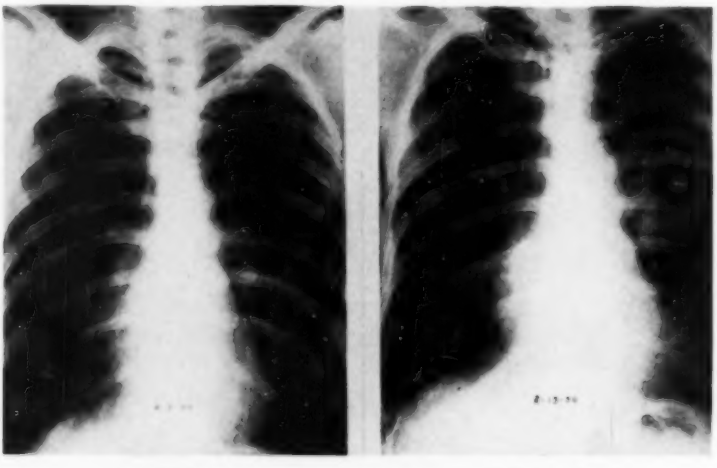

FIGURE 3A FIGURE 3B

Figure 3: Pulmonary Coccidioidomycosis. (3A) This is the initial roentgenogram which shows the lung abscess in the left upper lobe. The lesion is not yet cavernous. (3B) Eighteen months later, and after a period of healing, the left upper lobe lesion became a very large thin-walled cavity.

Six weeks after admission he became dyspneic, developed both right and left flank pain and complained of pleurisy on the left. Signs of right pleural effusion appeared and the x-ray film shown in Figure 1B was taken.

At this time a fragmented and friable lymph node was removed from the inferior border of the left pectoralis major and was reported: "lymph node showing

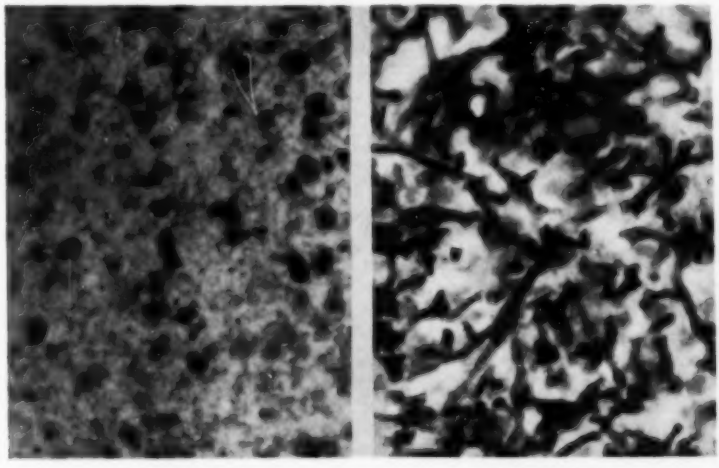

FIGURE 4A FIGURE 4B

Figure 4: Pulmonary Coccidioidomycosis. (4A) High dry magnification of an H and E. section. The field shown is in the wall of the cavity, close to its open surface. Little can be seen except caseation necrosis. (4B) The Schiff-stained section, with the same magnification, reveals a heavy network of mycelium, some of which is producing arthrospores. This is not the form expected in tissue.

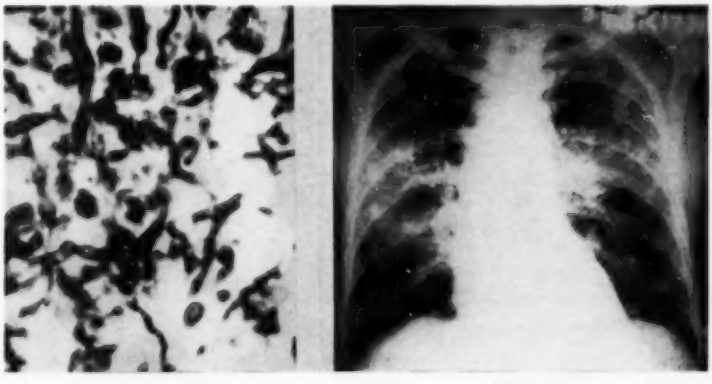

FIGURE 5 FIGURE 6

Figure 5: Pulmonary Coccidioidomycosis: Arthrospores in the caseous cavitary content. This is an oil-immersion photograph of an area in which the mycelium is almost entirely composed of arthrospores. Sputum from such a cavity would be highly infectious.—*Figure 6:* Pulmonary Histoplasmosis. This x-ray film was believed to be compatible with the pathologic diagnosis of Hodgkin's disease. It did not change significantly after treatment with nitrogen mustards. Histoplasma capsulatum was found in the pulmonary parenchyma at autopsy.

acute necrosis with granulomatous reaction. No acid-fast bacilli found on acid-fast stain." This was the first node in which Candida were later found by periodic acid-Schiff staining.

Thoracentesis revealed a straw-colored fluid which was negative culturally for pyogens and acid-fast bacilli. Subsequent specimens of pleural fluid were submitted both for culture and study for malignant cells and one specimen was cultured for pathogenic fungi without positive results being obtained by any of these procedures.

After the patient had received 42 grams of streptomycin he was discharged, but had to be readmitted within two weeks because of increasing dyspnea. His pleural effusion had increased and generalized lymphadenopathy was present. All nodes were tender as was the liver.

On this second admission he was afebrile during the first three weeks of hospitalization and febrile thereafter until the time of his death eight weeks after his second admission. An additional lymph node biopsy was done but it, together with further bacteriologic and microscopic studies of the pleural fluid, were unrevealing. The biopsy report read: "caseation necrosis compatible with tuberculosis." This was the second node to reveal foci of Candida in the areas of caseation necrosis. Nitrogen mustard did not influence his clinical course. Two weeks before his death he developed ascites, facial edema, and dysphagia. A culture of the sputum taken three weeks prior to death was reported as showing Candida sp. which was clinically considered to be a contaminant.

One week before death, during a severe paroxysm of coughing, the patient had a small hemoptysis accompanied by a 1 x 2 x 0.5 cm. of tissue which was saved and placed in formalin. This was sectioned and believed to show a neoplasm, type unspecified, probably lymphosarcoma. Permission for autopsy was not obtained.

Nine months after his death the lymph node biopsies were recut and stained by the periodic acid-Schiff method. As previously stated, two of these and the expectorated tissue were positive for Candida. Figure 2 is a central portion of the

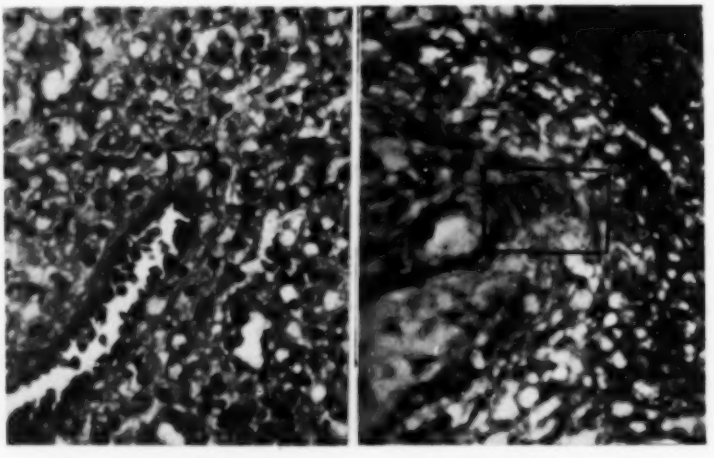

FIGURE 7A FIGURE 7B

Figure 7: Histoplasmosis. (7A) This photograph represents a high dry magnification of a portion of one of the lymph nodes biopsied in Shreveport and stained with hematoxylin and eosin. No organisms could be found under oil immersion. The area at the end of the blood vessel is reproduced in (7B). With the periodic acid-Schiff stain, numerous H. capsulatum could be found. The reticulo-endothelial cell at the base of the blood vessel is shown under oil immersion magnification in order to depict the large number of Histoplasma present.

expectorated tissue after it was restained by the periodic acid-Schiff method. The appearance of the fungus in tissue is so typical a diagnosis of moniliasis can be made although the species of Candida cannot be stated.

Figure 3 is a reproduction of two x-ray films of a middle aged white man who first became ill while residing in Arizona. The initial film was taken during a bout of "influenza" and was interpreted as showing an abscess in the left upper lobe probably coccidioidal in etiology. No fungi were isolated but the coccidioidin skin test was positive. By early 1949 this abscess was allegedly healed and the patient was asymptomatic. After returning to work, he became ill, and in September of 1949 a cavity was again found by x-ray film. At the time of his admission to Charity Hospital in November of 1949 he was producing between 500 and 1,000 milliliters of sputum daily, was febrile, and an x-ray film taken at that time showed a large left upper lobe cavity. Skin tests were negative through a 1:10 dilution of coccidioidin and complement-fixation and precipitin tests were reported as negative by Dr. C. E. Smith. The histoplasmin skin test was negative. Three sputum concentrates and one culture were negative for acid-fast bacilli and three sputa were negative on culture for pathogenic fungi. However, sputa cultured at the medical school laboratories were positive for Coccidioides immitis. A left upper lobectomy performed in March of 1950 was complicated by empyema. One month after operation a massive pulmonary hemorrhage occurred and the patient died.

The left upper lobe was submitted to the pathology department at the time of operation and was reported: "Lung abscess, chronic pneumonia, giant cell type. Bronchitis, chronic. There are numerous intracytoplasmic inclusions suggestive of virus inclusion. While giant cell pneumonia is not frequent in adults, it does occur rarely." The paraffin blocks were recut and sections submitted to periodic acid-Schiff stains. Figure 4 illustrates the findings by hematoxylin-eosin and periodic acid-Shiff staining. It is of special interest that sections through the wall of the cavity and extending into the cavitary content revealed the presence of arthrospores. An oil immersion photograph of some of these is shown in Figure 5. It is

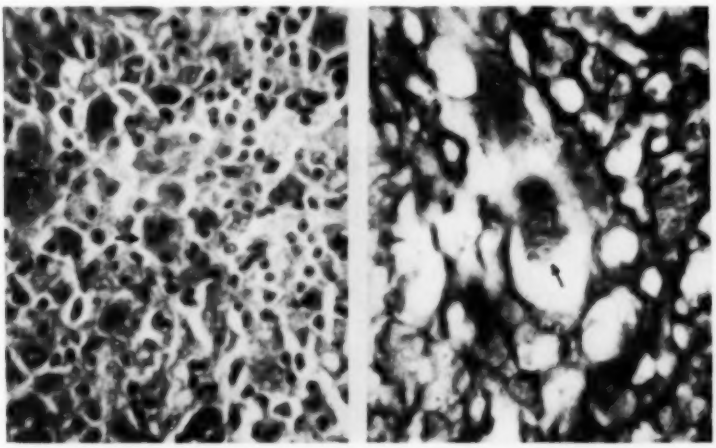

FIGURE 8A FIGURE 8B

Figure 8: Histoplasmosis. (8A) A portion of the first node to be examined at Shreveport showing typical granulomatous reaction with many Reed-Sternberg cells. This H. and E. section revealed no Histoplasma and all concurred in the diagnosis of Hodgkin's disease. The Dorothy Reed cell in the center of the field is shown in (7B) after periodic acid-Schiff staining and oil immersion magnification. It is stuffed with Histoplasma.

difficult to believe that fungi so obvious by the periodic acid-Schiff method could be so indefinite in sections stained with hematoxylin and eosin. The presence of arthrospores within the contents of the cavity is proof that this disease may be contagious. The size of the cavity afforded an environment more suitable to arthrospores than to the tissue-invading forms. Such circumstances must be quite unusual though one can now be sure that man to man transmission is possible.

The third patient was a 63 year old white farmer who was admitted to a hospital in Shreveport, Louisiana in May of 1951 complaining of increasing fatigability and general malaise of two years duration. He had experienced intermittent afternoon fever for 18 months and had lost 30 pounds in the preceding year. For several years he had been aware of a chronic nonproductive cough which had undergone no change. The admission examination was not enlightening except for the presence of prominent lymph nodes in the left axilla. These were hard but neither fixed nor tender. Sputum examination was negative for acid-fast bacilli and fungi on direct smear and one sputum was negative for Mycobacterium tuberculosis on culture. The chest x-ray film was interpreted as being within normal limits. While in the hospital his fever ranged between 100 and 103 degrees F.

On the seventh hospital day biopsy of the left axillary lymph nodes was undertaken and microscopic sections presented the picture of Hodgkin's disease. Smears and cultures of this lymph node were negative for acid-fast bacilli and pyogens. The patient was then transferred to a New Orleans hospital for irradiation therapy. His biopsy slides were reviewed and the diagnosis confirmed. He continued to be febrile, developed hepato-splenomegaly and supraclavicular lymphadenopathy. His total white blood cell count was 9,050 with a differential showing 93 per cent polymorphonuclear neutrophils.

Shortly after admission a left supraclavicular lymph node biopsy was obtained and was regarded as showing only marked necrobiosis with very few viable cells remaining. Those present revealed a granulomatous reaction which was considered nonspecific in the light of a negative acid-fast stain.

This patient was cyanotic, and extensive pulmonary pathology was revealed by

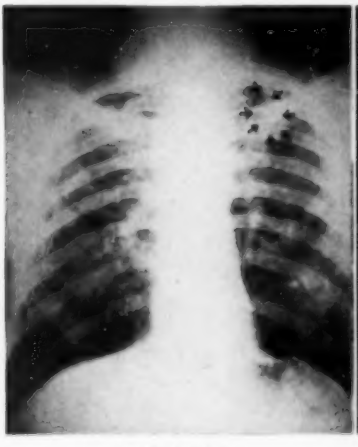

FIGURE 9

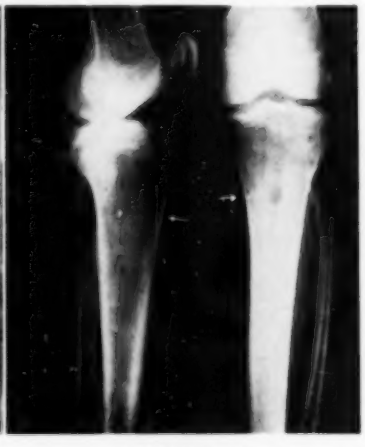

FIGURE 10

Figure 9: Pulmonary Blastomycosis. Extensive fibroproductive infiltration is present bilaterally and a 3 cm. cavity can be seen in the extreme apex on the left.—*Figure 10:* Blastomycotic Osteomyelitis. The two areas of osteomyelitis, one very small, show no reaction of the surrounding bone. The sequestra are very dense and small, simulating metallic fragments.

x-ray film as shown in Figure 6, and nitrogen mustards were given. Since there was no response to therapy, speculation arose concerning the possibility of this being something other than Hodgkin's disease. A third lymph node biopsy was then secured from the left axilla. Histopathology reported Hodgkin's disease with granulomatous areas with necrosis compatible with tuberculosis. Rare acid-fast rods were supposedly seen in a single section. All three biopsy specimens were

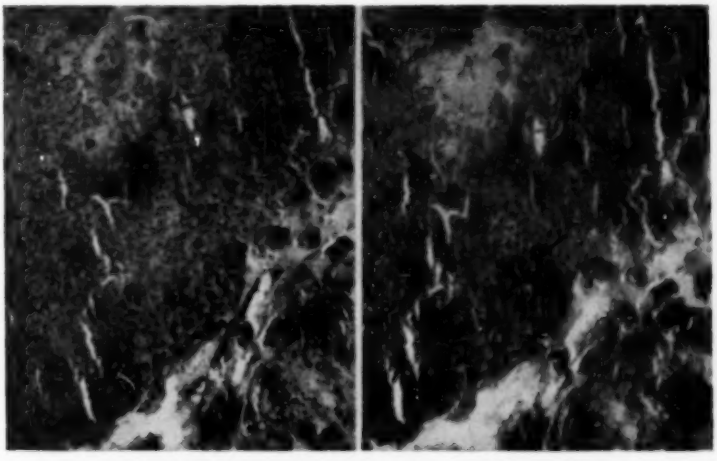

FIGURE 11A FIGURE 11B

Figure 11: Blastomycotic Osteomyelitis. (11A) The low-power H. and E. preparation reveals a chronic type of granulation tissue. (11B) The same field after staining with periodic acid-Schiff reagent. Note the rosette of Blastomyces in a giant cell.

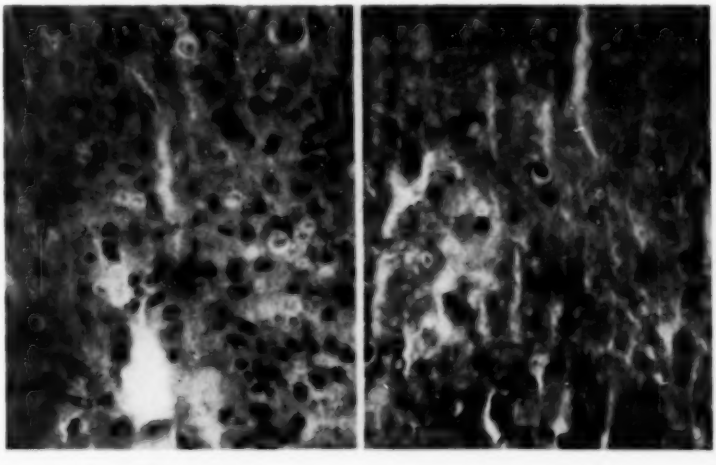

FIGURE 11C FIGURE 11D

Figure 11. Blastomycotic Osteomyelitis (continued). (11C) High-dry magnification of the H. and E. section shows bodies which are strongly suggestive of Blastomyces. (11D) Hotchkiss-McManus staining of same field. This is the same rosette seen in (11B).

then submitted to the Armed Forces Institute of Pathology which concurred fully with the diagnosis of Hodgkin's disease and tuberculosis.

In addition to the routine cultures for acid-fast bacilli, cultures on Sabouraud's dextrose agar and blood agar were obtained from the third biopsy. Fungus growth was noted by the eighth day and on the 11th day diagnostic tuberculate chlamydospores were seen and Histoplasma capsulatum recognized. No growth occurred on Petragnani's medium by the end of eight weeks. Very careful review of the previous hematoxylin and eosin stained slides failed to show any evidence of histoplasmosis. At this time permission was sought and gained to study the negative tissue blocks with the periodic acid-Schiff stain. Numerous Histoplasma capsulatum were demonstrated in all of the previously examined lymph nodes. In many areas most of the forms were degenerate and present largely as "ghosts"; in other areas perfectly typical and easily recognized intracellular forms were identified.

After Histoplasma were identified in the periodic acid-Schiff stained sections, it was not particularly difficult to locate them in the hematoxylin and eosin stained sections of the same node with the exception of the first nodes biopsied in Shreveport and the lymph node which revealed severe necrobiosis. In neither of these two biopsies were we ever able to recognize with certainty Histoplasma capsulatum in the hematoxylin and eosin preparations. Figures 7 and 8 illustrate the differences in appearance of these sections when stained by the two methods.

Additional positive cultures for Histoplasma were obtained from the bone marrow, blood, and sputum. Two weeks before his death an additional lymph node was removed from the left axilla and histological examination revealed a single focus of Histoplasma in the hematoxylin and eosin preparation. Hotchkiss-McManus stains of the same sections disclosed many organisms. Autopsy revealed generalized histoplasmosis.

The fourth patient was admitted to the Charity Hospital by transfer from another institution in New Orleans. This institution had made a diagnosis by x-ray film of tuberculosis and he was transferred to the tuberculosis unit of Charity Hospital. Ten months prior to admission he had suffered a "cold" with

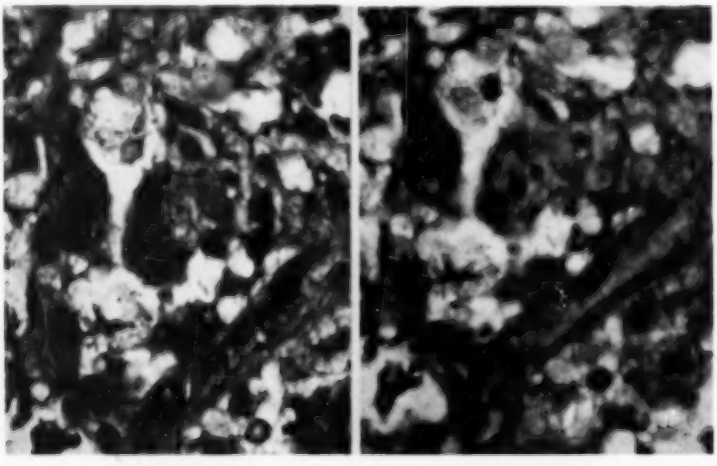

FIGURE 12A FIGURE 12B

Figure 12: Blastomycotic Lymphadenitis. (12A) The routine H. and E. stain revealed bodies which did not appear to be a part of a granulomatous reaction but their identity did not become apparent until the section was restained with the Hotchkiss-McManus stain (12B). Th two fields are identical.

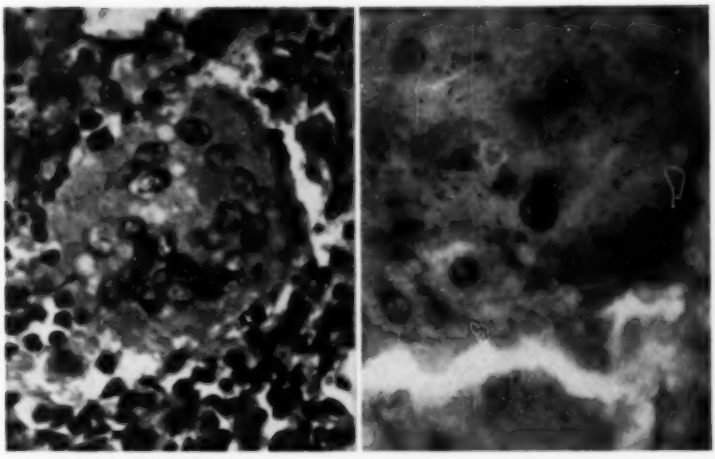

FIGURE 13A FIGURE 13B

Figure 13: Pulmonary Blastomycosis. (13A) In the multinucleate giant cell several round bodies, clear centrally, may be seen. This is a high-dry magnification of a H. and E. preparation. (13B) A marginal section of the multinucleate giant cell is shown with oil-immersion magnification after staining with the periodic acid-Schiff method. Note the budding Blastomyces.

FIGURE 13: Pulmonary Blastomycosis (continued). (13C) The planogram shows a cotton-ball density in the right lower lobe. Several lucencies are evident near the center of the lesion.

frequent remissions for short periods of time. Cough, productive of small amounts of white sputum had been present for approximately six months before his admission. Blood streaking of the sputum had occurred on two or three occasions. Hoarseness, which was progressive, began four months before hospitalization and became progressively more severe. Weakness, general malaise, and a 22-pound weight loss occurred over the six month period, but neither chills, fever, nor chest pain had been present. He was examined by his personal physician five months prior to admission. Physical examination together with a chest x-ray film revealed no abnormality. A family history of tuberculosis was present.

The first three sputum concentrates were negative for acid-fast bacilli, but the fourth was reported as positive. A Mantoux test was positive at 1:1000 dilution and the sedimentation rate was elevated. On the basis of the positive sputum concentrate, treatment for pulmonary tuberculosis was undertaken. Figure 9 is a reproduction of an x-ray film taken at this time. There were 16 subsequent sputum smears stained for acid-fast bacilli, all of which were negative. Eight additional sputum concentrates were negative. One sputum culture was negative for Mycobacterium tuberculosis and two cultures were negative for pathogenic fungi.

Laryngoscopy revealed an erosion with granulation tissue over the left true cord. Both cords were injected and inflamed. The larynx was similarly involved. It was the impression of the endoscopist that either laryngeal tuberculosis or carcinoma should be considered. Although there seemed to be some clearing of the chest x-ray film following treatment with bed rest, streptomycin and PAS, there was no change in the patient's hoarseness, weight, or general sense of well being. Because of this and the single positive sputum concentrate, laryngeal biopsies and cultural studies of the sputum for fungi were requested. The initial laryngeal biopsy was reported as showing: "Tissue from larynx showing chronic inflammation, with active lymph follicles." A specimen removed from the left vocal cord at the same time was reported: "Tissue from left cord showing epithelial hyperplasia with acute and chronic inflammation and scattered multinucleated giant cells with peripheral nuclei." Periodic acid-Schiff stains revealed many Blastomyces.

There was no change in his chest x-ray film after its initial improvement, or in his general course following additional therapy with Tibione and pneumoperitoneum.

Nine and a half months after admission he bumped his right leg against a bed-side chair producing a small hematoma. This was quickly followed by a draining sinus and the x-ray film shown in Figure 10 revealed two cystic lesions in the proximal tibial shaft. Two minute sequestra were evident. This was considered to be a tuberculous osteomyelitis and it was incised and drained and the pus cultured for Mycobacterium tuberculosis and pyogens. A portion of the curettings was sent to histopathology. Cultures of the pus were negative, and the curettings were reported by the pathologist: "Chronic debris. No acid-fast bacilli found." Figure 11 shows a hematoxylin and eosin stained section of these curettings and the same section when restained by the periodic acid-Schiff stain.

Thirteen months after admission a prominent and tender pretracheal lymph node appeared and was removed for biopsy. A granulomatous response with caseation necrosis was noted and the pathologist requested sections stained by the Ziehl-Neelsen method. In these sections he noted a few cyst-like bodies with a red staining central granule which were suspected of being Histoplasma. We were given a section of this node to restain by the periodic acid-Schiff procedure. Figure 12 illustrates a section of this node when stained with hematoxylin-eosin and when stained with the Schiff reagent. The periodic acid-Schiff stain of this node prompted us to review the previous biopsies and to culture the sputum for fungi. A laryngocutaneous fistula developed at the biopsy site and pus from this and the osteomyelitis in his right leg were likewise cultured for fungi. Sputum and pus from both areas were positive on culture for Blastomyces dermatitidis.

The last patient was a 56 year old negro hospital employee whose routine chest x-ray film prompted his admission for diagnostic studies. A rounded density was seen in the right lower lobe with no surrounding infiltration. The sharp margins suggested the possibility of a tuberculoma or neoplasm. The planogram which is shown in Figure 13 demonstrated several central lucencies and lung abscess was added to the differential diagnosis.

The patient was entirely asymptomatic and there was nothing of significance in the past history. The physical examination revealed no abnormality and the laboratory studies were within normal limits except for a positive skin test with old tuberculin. Sputum cultures for acid-fast bacilli, fungi, and pyogens were planted and smears were made and stained for malignant cells. Nothing of significance was found.

While these studies were in progress the patient was given large doses of penicillin without effect clinically or radiologically. Right lower lobectomy was performed without difficulty.

The lesion was found to have central ulceration and a portion of the lesion was ground in saline, centrifuged, and the immediately supernatant fluid cultured on blood agar and Sabouraud's dextrose agar. A portion of the material was likewise inoculated into the yolk sac of six chick embryos. Some of the original tissue was further ground and the tissue juice streaked on blood agar, Sabouraud's agar, and Petrik's medium. No fungi were isolated by any of these methods.

Hematoxylin and eosin sections of the embedded lung showed a very large number of organisms most of which were present within multinuclear giant cells. These were quite small, appeared encapsulated, and were thought to be Histoplasma capsulatum. A representative area from such a slide is shown in Figure 13A. Further study of the hematoxylin and eosin preparations lead to considerable doubt as to the nature of the fungus since budding forms were seen which looked more like Blastomyces dermatitidis or Cryptococcus neoformans than they did Histoplasma. The periodic acid-Schiff stain was then applied to this section as shown in Figure 13B. Typical thick-walled and budding forms were found, some of which were dumb-bell shaped with a heavy collar at the point of constriction. These forms were entirely typical of Blastomyces dermatitidis. However, it was of great interest that the majority of the forms did not stain well in the periodic acid-Schiff method. The staining of the walls appeared somewhat granular and slightly diffuse. Many, if not most, of the forms seemed to be undergoing some degeneration. Except for the beautiful demonstration of the budding and elongate forms which appeared quite normal, the general staining of the section by the periodic acid-Schiff method was inferior to that revealed by hematoxylin and eosin preparations.

Our embarassment at being unable to culture the fungus from the surgical specimen was lessened somewhat by the failure of the hospital laboratory with its numerous cultures of the same specimen. Skin tests with blastomycin were negative following operation. The patient was kept at bed rest without other treatment for four months and then discharged. There were no postoperative complications and no evidence of infection during the period of convalescence.

Although a final diagnosis is not possible without cultural proof, several of our colleagues as well as the Armed Forces Institute of Pathology were in agreement with the diagnosis of blastomycosis. One of us has previously noted that when Histoplasma capsulatum occurs for the most part in multinuclear giant cells, the forms are usually degenerating and either not viable or are culturable with great difficulty. It is possible that this phenomenon may be shared by Blastomyses and that failure in two laboratories to culture the organism is due to the small number of viable forms. There was much in the histopathology of this lesion to suggest that the body was overcoming the infection spontaneously. It is not unlikely that this lesion would have healed without any sort of treatment had it not been discovered by routine x-ray film.

Comment

It is true that with the exception of the first two cases presented the pathologists were able to retrospectively demonstrate the fungi by routine staining procedures when the area in which they were present was demonstrated by the Schiff stain. In the last case presented the organisms were more readily seen in the routine H and E preparation, but were initially misdiagnosed by one of us until their true nature became apparent after staining with periodic acid-Schiff reagent. It is our belief that anyone who will spend a few days learning the method and preparing a representative selection of slides will need no persuasion to convince him of its utility. Its primary advantage is that under low and high dry powers of magnification fungi can be detected which are completely missed by the usual low and high dry powers survey of the biopsy or autopsy specimen. With the staining methods in general use, fungi will be missed occasionally even when the sections are studied under the oil immersion objective. However, many hours can be spent in such a search without covering the area in which the agent is present if the infection is sparse. The color contrast of the periodic acid-Schiff stain immediately attracts one's attention to areas of suspicion and higher power observation will then confirm or disprove the presence of fungi in the area. One of the advantages of this stain is its applicability to sections which have been stained routinely months or even years before.

It is probably true that an etiologic diagnosis of mycotic infections can be made in many instances by using this staining method. However, this will not be generally acceptable, and rather than serving to decrease our efforts at cultural isolation and identification of the fungi, it should increase them.

A number of fungi may be found in sputum, bronchial exudate, or other body secretions and excretions without there being any certainty that their rôle is that of a pathogen. At times these fungi may be found within chronic abscesses or cavities in the lung or they may be seen in the alveolar exudate at postmortem examination. Whether such fungi are present as saprophytes or as pathogens may not be certain unless they can be demonstrated as tissue invaders. The periodic acid-Schiff stain is of special usefulness for this problem. For example, a patient dying from an aspiration pneumonia was found to have Candida in the alveolar exudate when stained with routine methods. A periodic acid-Schiff stain demonstrated these beautifully, but failed to show any evidence of tissue invasion.

SUMMARY

The periodic acid-Schiff stain has many uses, among them being the demonstration of fungi in tissue. Its use for this purpose is increasing but not as rapidly as it should. Besides revealing the fungi with exceptional clarity and detail, the factor of color contrast is very great. Organisms appear in varying shades of red and if light green is used as a background

stain, very rapid survey of stained sections is possible using the low and high power dry objectives.

Two cases are presented in which fungi could never be demonstrated by hematoxylin and eosin stains but were readily found by the periodic acid-Schiff method. Two additional cases are discussed in which the histological diagnosis was not made until biopsy material was stained with the Schiff reagent. A fifth case illustrates how proper identification may be greatly facilitated by this stain.

RESUMEN

El colorante periódico acido-Schiff tiene muchos usos entre ellos que sirve para la demostración de hongos en los tejidos Su uso con este fin está aumentando pero no tan rápidamente como debría. Ademas de que revela los hongos con claridad excepcional y con detalle, el factor de contraste de color es muy grande. Los organismos aparecen en tonos diversos de rojo y si el verde claro es usado como fondo de contraste es posible hacer una revisión rápida de los cortes coloreados usando los objetivos secos de bajo y alto poder.

Se presentan dos casos en los que los hongos nunca pudieron ser demostrados por la hematoxilina y eosina pero fueron facilmente encontrados por el método periódico acido-Schiff. Dos casos mas se discuten en los que el diagnóstico histológico no se hizo sino hasta que el material de biopsia fué teñido con el reactivo de Schiff. Un quinto caso ilustra cómo puede facilitarse grandemente la identificación adecuada por este colorante.

RESUME

La coloration acide de Schiff a beaucoup d'usages, parmi lesquels la possibilité de mettre en évidence les champignons dans les tissus. Son utilisation dans ce but es de plus en plus répandue, mais pas autant qu'elle le mériterait. En dehors du fait qu'ainsi se trouvent révélés les champignons avec une précision et une clarté exceptionnelle, il existe un facteur important dû au contraste. Les microbes paraissent dans différentes nuances de rouge, et si on réalise une coloration verte de fond, il est possible d'obtenir avec les objectifs secs à petit et à gros grossissement un examen très rapide des coupes colorées.

Les auteurs rapportent deux cas dans lesquels l'hématoxyline-éosine n'avait jamais pu mettre en évidence les champignons qui furent facilement révélés grâce à la méthode acide de Schiff. Deux observations dans lesquelles on ne put faire le diagnostic histologique avant que les coupes biopsiques n'eurent été colorées par le réactif de Schiff sont mises en discussion. La cinquième observation montre comment une identification précise peut être largement facilitée grâce à cette coloration.

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Management of the Patient Dying of Bacterial Pneumonia*

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Patients with bacterial pneumonia usually recover promptly when given modern antibiotic preparations. Choice of the antibiotic for each case continues to be discussed as the major responsibility of therapy. Reported mortality rates range from 5.7 to 9.3 per cent.[1] Deaths occurring with this frequency from infectious processes indicate an opportunity for improving treatment.

In the period from 1948 to 1951 nine patients died from bacterial pneumonia, during which time 163 cases were admitted. The uncorrected mortality rate was 5.5 per cent. The records of fatal cases were analyzed and the following reports indicate some of the problems encountered in patients who die under hospital management.

Case 1 (248043): A white female, 44 years of age, was admitted on the third day of pneumonia with temperature of 101.8 degrees F., pulse 128 and respirations 24 per minute. The lower lobe of the left lung was consolidated. The blood pressure was 149/82 mm. Hg. Total white blood count was 23,600. The patient was known to have myasthenia gravis. Treatment included prostigmine, oxygen, penicillin, and sulfadiazine. Death occurred four days after admission. Lung drainage in this patient was likely inadequate because of deficient muscular force.

Case 2 (560769): A colored male foundry worker, aged 41, was admitted in a stuporous state. He had had a mild cold for three weeks. Nausea, vomiting, and abdominal pain began five days before admission. The temperature was 102.8 degrees F., pulse 140 and respirations 32 per minute. The blood pressure could not be obtained. Moist rales were present at both bases. The white blood count was 6,.400. Death occurred in three hours. The admission blood culture revealed pneumococci, type VII. Postmortem examination revealed consolidation of the right middle and both lower lobes. The adrenal glands showed "toxic" changes and lipoid depletion. He was given oxygen, intravenous fluids, and the correct antibiotic. Peripheral vascular collapse was not corrected.

Case 3 (175857): A male, aged 57, had a chill two days before admission which was associated with vomiting, diarrhea, bloody sputum, and pain in the right chest. He was mentally clear on admission. The temperature was 101.6 degrees F., the pulse 110 and respirations 26 per minute. The blood pressure was 80/50 mm. Hg. The upper lobe of the right lung was consolidated. White blood cell count was 1,850. Sputum and blood culture revealed Klebsiella pneumoniae. Treatment included fluids, intravenous glucose and whole blood, streptomycin, sulfadiazine and ephedrine sulfate. The patient expired in 29 hours. At autopsy both adrenals showed hemorrhagic necrosis.

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†Deceased, June 15, 1953.

Case 4 (596615): A delirious obese white male, aged 58, was admitted with a fever of 105.4 degrees F., a pulse rate of 140 and respirations of 48 per minute. The blood pressure was 130/90 mm. Hg. Cyanosis was present. Consolidation was found on the right side of the chest. The white blood cell count was 2,050. Streptococcus viridans grew in the admission blood culture. He was given complete standard therapy and nursing care. With a decline in blood pressure, he was also given ephedrine sulfate without effect. He expired 25 hours after admission. The right upper and middle lobes were shown to be consolidated at postmortem examination. The pathology included toxic changes in liver, heart, kidneys and brain. Vacuolization and antemortem cloudy swelling was present in the adrenals.

Case 5 (226610): A delirious white male, aged 52, was admitted on the fourth day of an illness evidenced by dyspnea, fever, chills and pain in the left chest. Admission temperature was 104 degrees F., pulse 160 and respirations 30. Bloor pressure was 50/30 mm. Hg. The chest was barrel shaped, cyanosis extreme, and respirations bubbling. The white blood count was 10,500. Type III pneumococci were present in the sputum. He was given fluids, oxygen, antibiotics, and 15 cc. of whole cortical extract. Death occurred in eight hours. Postmortem findings were fibrinous pericarditis and pleuritis and consolidation of the left lung with areas of necrosis. The adrenal glands were hypertrophic with necrosis of the zona fasiculata and an ill defined zona glomerulosa.

Case 6 (350196): A white male, aged 70, was admitted the day following sudden right pleural pain, nausea and vomiting. His temperature was 101.2 degrees F., pulse 140 and respirations 40 per minute. Systolic blood pressure was 48 and diastolic 25 mm. Hg. A friction rub was present in the right axilla. The initial diagnosis was pulmonary infarction. The sputum revealed type III pneumococci as did the blood culture. Antibiotics were not given until 11 hours after admission. Death occurred 12 hours later. Consolidation of the right lung was revealed at autopsy.

Case 7 (596998): A white female, aged 65, known to have arterial hypertension and diabetes mellitus was admitted in a stuporous state two days after the onset of diarrhea, vomiting and coughing. Temperature was 101.2 degrees F., pulse 120 and respirations 24. The white blood count was 700 with no polymorphonuclear leucocytes seen. She was given penicillin and streptomycin on admission. The diabetes mellitus was well controlled. Consolidation of the upper lobe of the right lung developed. Death resulted in 44 hours. Organisms present in the lung at autopsy were escherichеae coli. Numerous acute ulcerations were present in the colon. The spleen showed a myeloid metaplasia.

Case 8 (496714): A white male, aged 64, was admitted in a moribund condition after a five day illness at home. Consolidation of all lobes on the right was noted. The systolic blood pressure was 110 and diastolic 80 mm. Hg. He was given penicillin but died in three hours with pulmonary edema and peripheral vascular collapse. Postmortem examination was not allowed.

Case 9 (560618): A male, aged 64, who developed chest pain, dyspnea, and cough productive of bloody sputum, was admitted three days after onset. He was comatose on admission. He had been given penicillin, streptomycin and a sulfonamide at home. The upper lobe of the left lung was consolidated. The white blood cell count was 5,200. With unsatisfactory response to standard therapy, temperature reached 106 degrees F., peripheral vascular collapse ensued. The patient expired 22 hours after admission without regaining consciousness. Thirty mgms. of cortisone was given a few minutes before death. Postmortem examination revealed consolidation of the left upper lobe, pulmonary emphysema, and a bone marrow fibrosis designated as agnogenic myeloid metaplasia.

Six of the nine patients who died had important associated conditions. In two pulmonary emphysema was present. Myesthenia gravis, obesity, diabetes mellitus and agranulocytosis, and agnogenic myeloid metaplasia, were individual handicaps in four others. Other pertinent data from these nine cases are summarized in Table I.

Patients admitted to the hospital at present are chiefly those with important associated handicaps or those who have failed to respond to the variable but usually effective antibiotic treatment given at home. In these patients standard therapy of oxygen, fluids, relief of pain, and further antibiotics will frequently prove inadequate. Prompt isolation of the etiological organism and the use of specific antibiotics will not regularly avert death. This is especially true in the aged, or in those having alcoholism, diabetes mellitus, obesity, cardiac or pulmonary ventilatory insufficiency. A judgment of impending death may be made from the type of onset, and

TABLE I: FATAL BACTERIAL PNEUMONIA

Age:		
40-49	2	
50-59	3	
60-69	3	
70-79	1	
Clinical State:		
delirium or coma	6	
peripheral vascular collapse	5	
restricted respiration	4	
vomiting	4	
Bacteriology:		
pneumococcus, type III	2	
pneumococcus, type VII	1	
Klebsiella pneumoniae	1	
Streptococcus viridans (blood)	1	
Escherichieae coli (agranulocytosis)	1	
not determined	3	
TOTAL		9
White Blood Count:		
Less than 4,000	4	
4,000 to 10,000	2	
10,000 to 20,000	1	
23,000	1	
not determined	1	
TOTAL		9
Adrenal Glands:		
Hemorrhage	2	
Cortical damage	3	
Normal adrenal	2	
Not determined	2	
TOTAL		9

the clinical features noted on admission. Table II outlines data related to body systems which are of value in judging the immediate future of the patient.

Standard and supportive treatment and nursing care has not prevented death in terminal cases judged as such on admission. If the emergency is recognized and supplemental treatment given, experience indicates that hospital mortality rates in bacterial pneumonia can be further reduced. The following case abstracts report the type of treatment which has appeared to prolong life sufficiently to allow the infection to be controlled.

Case 10 (670148). A male, aged 77, was brought to the hospital because of acute disorientation preceded by five days of non-productive cough. Respiratory grunting was present with a temperature of 103 degrees F., pulse 84 and respirations of 18 per minute. The patient had an advanced pulmonary emphysema. Systolic blood pressure was 135 and diastolic 75 mm. Hg. The white blood cell count was 16,000. Radiological examination revealed central lung consolidation on the right. The patient was given standard therapy which included 400,000 units of penicillin and 0.5 gram of streptomycin. Five hours later the patient was moribund, auricular fibrillation was present and the systolic blood pressure was 78 mm. Hg. He was given 25 mgm. of cortisone and 5 cc. of adrenal cortical extract intramuscularly together with 15 cc. of the exartct intravenously. Ten milligrams of desoxyephedrine was administered both intravenously and intramuscularly. Cedelanid and 1.5 million units of penicillin were given intravenously. Within 30 minutes he was conscious, comfortable and his systolic blood pressure was 105 mm. Hg. Cortisone was thereafter given orally every six hours in 25 mgm. doses. This was gradually reduced and was discontinued five days after admission when 20 mgm. of ACTH was given in one dose. He had no fever 12 hours after supplemental treatment was started. No extension of disease occurred, and clearing as shown by x-ray films was complete.

Case 11 (663847): A 78 year old factory worker was brought to the emergency room delirious and raising rusty sputum. One week before admission he "caught

TABLE II: IMMEDIATE PROGNOSIS POOR

Systems:	
Central nervous	onset delirium convulsions coma
Gastro-intestinal	onset distension vomiting dehydration
Respiratory	limited motion frequency above 40 substernal pain
Cardiovascular	hypotension rate exceeding systolic blood pressure auricular fibrillation
Hematopoietic	white blood count below 4,000

a chest cold" but continued working until 12 hours before admission at which time he had developed nausea, vomiting and confusion. His temperature was 103 degrees F., pulse 100 and respirations 46 per minute. The blood pressure was 90/40 mm. Hg. Cyanosis was present and the upper lobe of the right lung was consolidated. The sputum smear revealed many gram-negative bacilli and culture contained N. catarrhalis and non-hemolytic streptococci. He was given oxygen, glucose, and 1,000,000 units of penicillin intravenously immediately together with 400,000 units intramuscularly. After the sputum smear was examined 500 mgm. of aureomycin was given intravenously. This was repeated after six hours. Cortisone was started, at 15 mgm. intramuscularly every two hours. The mental state improved but the blood pressure remained at 70/60 mm. Hg. Fifteen milligrams of desoxyephedrine was given intramuscularly. Within 30 minutes the blood pressure was 100/70 mm. Hg. Twelve hours after admission he was alert, oriented and had a good appetite and color. Cortisone (25 mgm.) and aureomycin (500 mgm.) were then given every six hours by the oral route. The cortisone was gradually reduced and was discontinued on the fourth hospital day. No extension of the lung consolidation occurred and resolution was usual. Blood cultures remained negative.

Case 12 (52725): A white woman, aged 71, was admitted in a confused dehydrated state, two days after the onset of vomiting, cough and left chest pain. The initial temperature was 99 degrees F. The upper lobe of the left lung was consolidated. When she reached her hospital room the temperature was 103 degrees F., pulse 120 and respirations 24 per minute. Blood pressure was 130/60 mm. Hg. The sputum smear revealed pneumocicci, type III. The white blood count was 13,900. Oxygen, fluids and 2.5 million units of penicillin were given. A special nurse was engaged. After seven hours the temperature was 103 degrees F., pulse 110 and respirations 44 per minute. In the next 12 hours she received 75 mgm. of cortisone and 2.5 million units of penicillin. Blood pressure remained at 100/45 mm. Hg. Eight hours after beginning cortisone she was afebrile, alert and without cyanosis or dyspnea. Blood cultures remained sterile. Cortisone was discontinued after 48 hours.

Case 13 (588094): A 58 year old man was admitted with chills, fever, chest pain and bloody sputum on the fourth day of illness. He had mitral stenosis, pulmonary emphysema and mild congestive heart failure. Digitalis was being taken. The temperature was 103.4 degrees F., pulse 110 and respirations 40 per minute. The blood pressure was 140/60 mm. Hg. The left upper and lower lung lobes were consolidated. The white blood count was 10,400. The sputum contained Klebsiella pneumoniae. Initially he was given oxygen, codiene, aminophyllin, glucose solution intravenously and aureomycin orally. In two hours he was moribund with bilateral respiratory bubbling. The blood pressure was 110/50 mm. Hg. He was then given 100 mgm. of cortisone by mouth and thiomerin and streptomycin by injection. Within three hours the temperature was 100.4 degrees F., blood pressure 140/70 mm. Hg. and cyanosis and dyspnea were decreased. After five additional hours he was comfortable and free of fever; blood pressure was 130/80 mm. Hg. Cortisone was discontinued on the fourth day. Recovery was uneventful.

Since observing the symptomatic effects of nebulized cortisone in the treatment of bacterial pneumonia, a total of 11 patients have been given adrenal cortical supplements during the initial period of therapy. In three patients the medications were given although the clinical state did not appear to be a terminal one. In seven patients steroid hormones were administered because of an appearance of impending death. One obese patient with delirium tremens and lobar pneumonia was given ACTH. In conjunction with antibiotics, blood cultures remained sterile, no spread of

infection to other lobes occurred and no complications were observed. The 11 patients recovered.

Standard therapy has not been omitted. Antibiotics have been administered in adequate doses, preferably by the intravenous route in patients in circulatory collapse. For dehydrated patients intravenous fluids may be necessary in large amounts. One patient received 5 liters of fluids in the first 12 hours. Hypotension persisting after adequate hydration may be combatted with pressor amines. Desoxyephedrine hydrochloride ("Drinalfa") has been effective in most cases in doses of 15 to 20 mgm. every three hours as necessary. Oxygen can be delivered to the patient by tent, mask, or nasal catheter, depending on the circumstances. Attention to lung drainage is essential. The patient with chronic pulmonary disease is severely handicapped when he contracts pneumonia. Those with emphysema, bronchiectasis, pulmonary fibrosis, or asthma, besides the handicap of reduced pulmonary function, have poor lung drainage. In these people, pus and mucus accumulate and add to hypoxemia. Dramatic results have been obtained with passage of an endotracheal tube with repeated aspirations of the trachea. Paroxysmal auricular fibrillation has responded well to intravenous lanatoside C ("Cedelanid").

Consideration of the previously autopsied cases where five out of the seven showed structural evidence of damage to the adrenal cortex, allows speculation about the value of adrenal cortical supplements as adjuncts to treatment. Rich[2] in 1944 suggested a relationship between circulatory collapse in fulminating acute infections and adrenal cortical damage. The poor response of Addisonian patients to stress of infection is well known. Patients who have an excess of adrenal cortical hormones (Cushings Disease) also react poorly to infection. However, the aim in treatment in the patients described in this paper is supportive. In patients in whom daily eosinophil counts have been performed, no rise above normal levels was seen after termination of cortisone. This may possibly indicate that a hyper-adrenal cortical response was not produced. The purpose of the suggested adrenal supplements is the temporary preservation of life until such time as other measures become effective. The administration of cortisone or adrenal cortical extract is followed promptly by a lessening of delirium and vomiting, by improved respiratory function and by a recovery of blood pressure. The patient becomes comfortable, cooperative, takes fluids and breathes easily. ACTH has been used rarely because so large a percentage of our autopsied cases showed adrenal damage. It is not known whether exogenous ACTH would harm adrenal glands possibly already failing from the stress of overwhelming infection.

ACTH and cortisone have been used by others in acute infections. Perla and Marmorston[3] in 1940 treated pneumonia with small doses of adrenal cortical extract, and believed that blood pressure was better maintained, "toxicity" was decreased, appetite and sense of well being were improved. Reeder and Mackey,[4] from this hospital, reported prompt symptomatic improvement after inhalation cortisone. Kass, Ingbor and Finland[5] used ACTH in pneumococcal lobar pneumonia. In typhoid, Smadel et al.[6] and

Woodward et al.[7] used cortisone to advantage. In peritonitis, Rapeno et al.[8] and Boling et al.[9] found ACTH or cortisone in conjunction with antibiotics to give a better result than antibiotics alone.

Cortisone has been given initially intramuscularly in doses of 50 to 100 mgm. Patients are able to take 25 to 50 mgm. orally every six hours after the first intramuscular dose. After 24 hours it has been possible to gradually decrease the dose, and terminate it after three or four days. The use of adrenal cortical extract has been reserved for those patients with peripheral vascular collapse either at the start of treatment or after the pressor amines have failed to elevate the blood pressure. Adrenal cortical extract has been used in doses of 20 to 40 cc. every six hours intravenously. In the cases when this drug was used, it was possible to stop this medication after one or two doses.

Known effects of the steroid hormones on the tissue reaction to infection furnish theoretical objections to their use in bacterial pneumonia. The present study revealed no injury to tissue or to patient resulting from the use of these agents. In conjunction with antibiotics and other measures, blood cultures remained sterile, no extension of infection to other lobes occurred, no complications were observed, and recovery was uneventful.

SUMMARY

Of 163 consecutive cases of bacterial pneumonia nine died. Seven of these were autopsied and in five adrenal damage was present.

Pressor amines and adrenal cortical supplements have appeared to maintain life until body processes and antibiotics have controlled the infection.

RESUMEN

Entre 163 casos consecutivos de neumonía bacteriana, murieron nueve. Siete de estos, fueron autopsiados y en cinco, se encontró daño de las suprarrenales.

Parece que las aminas presoras y los suplementos adrenocorticales, ayudan a mantener la vida hasta que los procesos defensivos del cuerpo y los antibióticos, dominan la infección.

RESUME

Les auteurs ont noté neuf décès sur 163 cas consécutifs de pneumonies à bactéries. Sept parmi ces décès furent autopsiés, et dans cinq cas on constata l'existence d'altérations des glandes surrénales.

Les amines hypertensives et l'apport d'extraits de corticosurrénales ont semblé maintenir le malade en vie, jusqu'à ce que l'infection ait été jugulée par les processus de défense et les antibiotiques.

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Segmental Resection in Pulmonary Tuberculosis*

(A Preliminary Report)

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Rapid strides have been made in thoracic surgery during the past 15 years due to the improvement of operative technique, the development of modern anaesthesia and the discovery of new antibiotic agents. In the early years little enthusiasm was aroused for pulmonary resection in tuberculosis because of the high operative mortality and morbidity. Through the efforts of many workers, such as Churchill and Klopstock,[1] Thornton and Adams,[2] Overholt et al.,[3-7] Sweet[8,9] Bailey,[10] Sarot[11] and others, resection in pulmonary tuberculosis has become an established method of treatment. At the present time, the surgical technique has been fairly well standardized and resection can be considered a reasonably safe procedure.

However, the aim of pulmonary resection in tuberculosis has been not only to remove the most evident lesion, but also all foci which may eventually contain tubercle bacilli. Often, pneumonectomy has been performed instead of planned lobectomy because at operation an incomplete interlobar fissure was found, or because the tuberculous process extended across the fissure to involve the adjacent lobe. The tendency has been to remove too much rather than too little tissue; surgeons have been too radical in the resection of lung parenchyma.

Increased experience in lung tissue resection has demonstrated that the bronchopulmonary segment is a surgical as well as an anatomical unit, and can be removed without undue difficulties. Exact techniques of segmental resections have been elaborated by Churchill and Belsey,[12] Clagett and Deterling,[13] and particularly by Overholt and Langer[14] who eliminated the use of clamps on lung tissue and so avoided damage to healthy segments. This operative procedure, while widely used in the surgical treatment of bronchiectasis, for many years has been almost prohibited in excision therapy of pulmonary tuberculosis for fear of transecting lung parenchyma in the neighborhood of a tuberculous process. The experience of the last few years, however, has demonstrated that the separation of a diseased lobe from the adjacent lobe by blunt and sharp dissection or, when separation was technically impossible, by removal of a slice of parenchyma did not result in local spread or exacerbation of the disease. This encouraged many surgeons to attempt the removal of segments containing evidently offending lesions and to preserve lung tissue which was, on palpation during operation, free of disease.

The encouraging results of segmental resection in pulmonary tubercu-

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losis reported by M. Chamberlain and Klopstock[15] in 1950 induced us, not without hesitation, to try this procedure on a series of patients that seemed to us suitable for limited excision. This preliminary report deals with the indications, the immediate results, the operative mortality and the incidence of morbidity in 36 segmental excisions in pulmonary tuberculosis performed by us from March 1950 to July 1951.*

Material and Indications

The patients have been selected at combined medical and surgical conferences in the various institutions of the country, and always with the participation of our leading phtisiologists. Each case has been broadly discussed before decision was made. The patient was observed clinically for a few months prior to admission for surgery. Thorough and repeated x-ray studies, particularly lateral tomograms for exact localization of the involved segment, have always been presented at the conferences.

The main indications for segmental excision as employed in Group 1 and presented in Table I, have been in patients with a stabilised disease where the offending focus has been confined to one or two segments of a lobe, after a period of strict bed rest between three and six months. There were patients who, in view of the nature of the disease or its location, were unsuitable for collapse therapy or would require an extensive thoracoplasty with rather doubtful results (see Figures 1A, 1B and 1C). In some instances there was more than one indication, but we have tried to classify them according to the indication of primary importance. There were 12 cases of solitary or multiple nodular infiltrates with distinct areas of rarefaction suggesting small cavities (see Figures 2A, 2B and 2C). Some of them were authentic tuberculomata, others were poorly encapsulated caseous foci or inspissated cavities. In six cases the indication for resection was an atelectatic segment of a lobe with one or several cavities. On eight occasions the operation was performed for disease of the lower lobe (see Figures 3A, 3B and 3C; 4A, 4B and 4C).

In Group II segmental resection was done in preference to thoracoplasty.

*Since this paper has been presented 92 additional segmental excisions in pulmonary tuberculosis have been performed.

TABLE I: INDICATIONS

		Number of Cases
Group I		26
A) Tuberculoma, nodular infiltrates with areas of rarefaction	12	
B) Atelectatic segments with cavities	6	
C) Lower-lobe disease	8	
Group II:		
In preference to thoracoplasty		2
Group III		
Disseminated predominantly unisegmental disease		8
	TOTAL	36

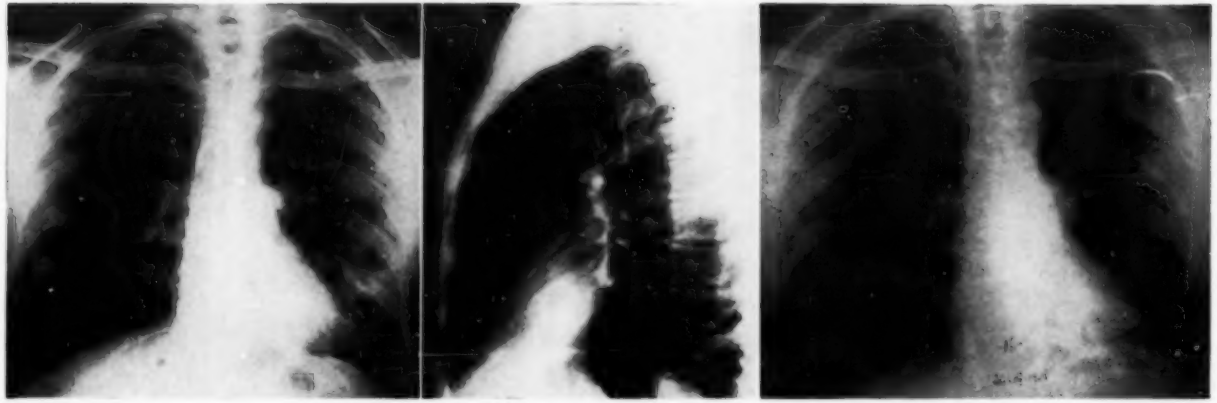

FIGURE 1A FIGURE 1B FIGURE 1C

Figure 1A: P.J., Male, 29-year old diabetic patient with a cavity in the lingula of the left upper lobe, an infiltrate in the superior segment of the left lower lobe and another in the right upper lobe.—*Figure 1B:* P.J. The lingular cavity in lateral view.—*Figure 1C:* After unsuccessful treatment with left phrenic crush and 60 grams streptomycin, excision of the ligula and the superior segment of the lower lobe was performed.

This group includes two patients with diseases which might have been controlled by means of thoracoplasty. Resection was performed upon the request of the patients and the insistence of the treating physicians.

In Group III the indication for segmental resection was one of the most controversial subjects. Whereas in the formed groups the disease was preoperatively considered to be localized in a segment of a lobe, in this group there were patients with multilobar and sometimes bilateral disseminated disease (see Figures 5A, 5B and 5C). The most offending foci, however, were predominantly unisegmental and confined to one lobe. In most of these, thoracoplasty was either contraindicated because of the presence of contralateral disease, or it was anticipated that thoracoplasty would fail to control the disease in view of its location. In our opinion there was nothing else to be offered to these patients. Our aim was to remove the worst of the tuberculosis, thereby putting the patient in a more favorable condition to control the remainder of the pathology. There were eight patients in this group

General Statistics

Thirty-six operations have been performed on the same number of patients (see Table II) of whom 20 were between 21 and 30 years of age. All had positive sputum for tubercle bacilli. This was one of the required conditions to be admitted for segmental resection. None at preoperative bronchoscopy, had endobronchial disease of the major bronchi. Fourteen operations were done on the right and 22 on the left lung. As pointed out by Chamberlain and Klopstock,[16] the most frequently involved segments of the lung were the apical and posterior segments of the upper lobe and the superior segment of the lower lobe. We have on five occasions resected the apical segment, on seven the posterior segment, and in seven patients both the apical and the posterior segments of the upper lobe. The superior segment of the lower lobe was resected in six patients and in two additional cases together with the apical and the posterior segments of the upper lobe. In brief (see Table III) the apical segment was involved in 14 cases, the posterior in 16 and the superior segment of the lower lobe in eight. Whenever the process was localized to a part of a segment a subsegment was resected after pain-staking dissection of its bronchus and vessels. We have on eight occasions resected isolated subsegments. In 10 persons two segments have been removed and in one patient three segments from two adjacent lobes. No wedge resections have been done.

Five had been preoperatively classified as poor surgical risks and 31 as reasonable risks. Two had severe diabetes, and one essential hypertension with a history of "coronary attacks."

Twenty-five were admitted for surgery with strictly unilateral disease. In four, calcified foci or old scars in the contralateral lung had been demonstrated radiologically. Seven had evident contralateral disease or recent bronchogenic spreads which subsided during the preoperative sanatorium observation.

Ten had previous unsuccessful ipsolateral artificial pneumothorax and

FIGURE 2A FIGURE 2B FIGURE 2C

Figure 2A: S.I., Female, aged 29, presenting an old silent process in the left upper lobe of five year duration at least, had a flare-up in October 1959, with a high temperature and tomographic appearance of multiple round foci in the posterior segment (7 cm.) of the upper lobe, and *Figure 2B:* A paramediastinal cavity in the anterior segment (12 cm.) of the same lobe.—*Figure 2C:* S.I., after four months' sanatoria treatment, the anterior and the apicoposterior segments were resected.

TABLE II: GENERAL STATISTICS

1) *Number of Patients*			36
Right Lung		14	
Upper Lobe			
Apical segments	3		
Apical subsegment	1		
Posterior segment	1		
Posterior subsegments	2		
Apical and posterior segment	1		
Apical and posterior subsegment	1		
Apical and posterior and superior segment of lower lobe	1		
Lower Lobe			
Superior segments	3		
Posterior basals and lateral basal	1		
Left Lung		22	
Upper Lobe			
Apical-posterior segment	5		
Apical subsegment	1		
Posterior subsegments	4		
Apical-posterior and anterior segment	5		
Apical-posterior and superior segment of lower lobe	1		
Lower Lobe			
Superior segments	3		
Superior segmnet and posterior basal	1		
2) *Age of Patients (15 to 51 years)*			
15 to 20	5		
21 to 30	20		
31 to 40	8		
41 to 51	3		
3) *Sex*			
Male	19		
Female	17		
4) *Incidence of Positive Sputum*	100 per cent		

TABLE III: THE MOST FREQUENTLY INVOLVED SEGMENTS

1) Posterior segment, upper lobe	16
2) Apical segment, upper lobe	14
3) Superior segment, lower lobe	8

TABLE IV: PREVIOUS THERAPY

	Ipsalateral	Contralateral
Artificial pneumothorax	10	2
Pneumoperitoneum	1	1
Phrenic nerve crush	1	
Thoracoplasty		1

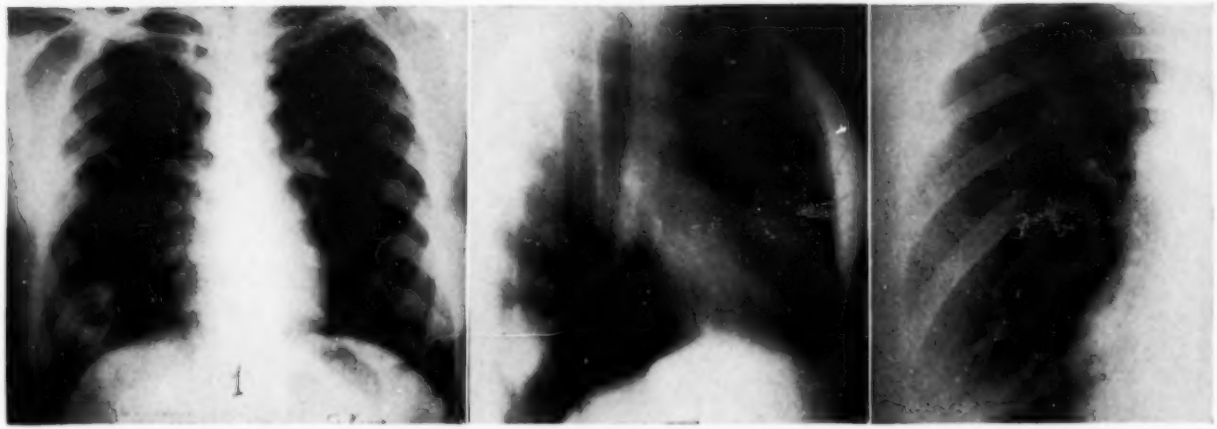

FIGURE 3A FIGURE 3B FIGURE 3C

Figure 3A: M.R., Female, aged 22 years, with a cavity in the right lower lobe, unsuccessfully treated with artificial pneumothorax from March 1949 to November 1950.—*Figure 3B:* M.R. The morbid condition localized in the basal posterior segment, on lateral tomogram.—*Figure 3C:* M.R. The basal posterior and lateral segments were resected on January 22, 1951.

one a phrenic nerve crush (see Table IV). One had contralateral effective pneumothorax while she was admitted for surgery. One had been treated by artificial pneumoperitoneum and one had contralateral seven rib thoracoplasty 18 months prior to the segmental resection (Figures 6A, 6B and 6C).

Fourteen patients have been treated by systemic administration of streptomycin in the past, and some of them were given PAS.

Technical Considerations

We have adopted Overholt's face down position as a routine for lung excision in tuberculosis. We found, however, that the segments of the upper lobe are more easily approached in the lateral position. We have therefore been using the lateral position in all lesions of the upper lobe from which minimal drainage of secretions was anticipated. Twenty-one operations were done in Overholt's position and 15 in the lateral position. Application of clamps on lung parenchyma has been avoided in segmental resection, and no pleuralization of the row area by free grafts nor approximation of the raw surfaces by means of sutures is attempted as a rule. In some cases, however, when a small wedged space remained after removal of a segment, the pleural surfaces were occasionally approximated without interference with the complete expansion of the remaining lung tissue. Very often, however, small hematomata persisted in the intersegmental plane for several weeks, and therefore we try to eliminate approximation.

In the majority of cases the intersegmental veins have been visualized and left on the remaining segment. If, however, the focus to be removed is very close to the healthy segment, the veins and occasionally a slice of the adjacent segment are resected.

Complications

We had neither major complications nor fatalities in our first 21 consecutive segmental resections. In the whole series we had no operative spreads, no local exacerbations in the intersegmental plane, no flare-up of existing lesions in the remaining lobes or in the contralateral lung. There was complete expansion of the lung with obliteration of the pleural cavity during the first few postoperative days. Some patients developed small apical or intersegmental hematomata which gradually underwent resorption or organization in the first few weeks following surgery.

Two patients in whom the apico-posterior and the anterior segments of the left upper lobe were resected developed bronchopleural fistulae with small apical empyemata. Both promptly recovered after simple tube drainage of the empyema. The fistulae closed spontaneously with complete obliteration of the apical pocket after re-expension of the lung.

Mortality

There were two fatalities in the last 15 cases.* One died of anuria subsequent to a transfusion given during the operation. He expired six days after

*There was no mortality in the additional 92 segmental resections. There were therefore two fatalities in 128 operations.

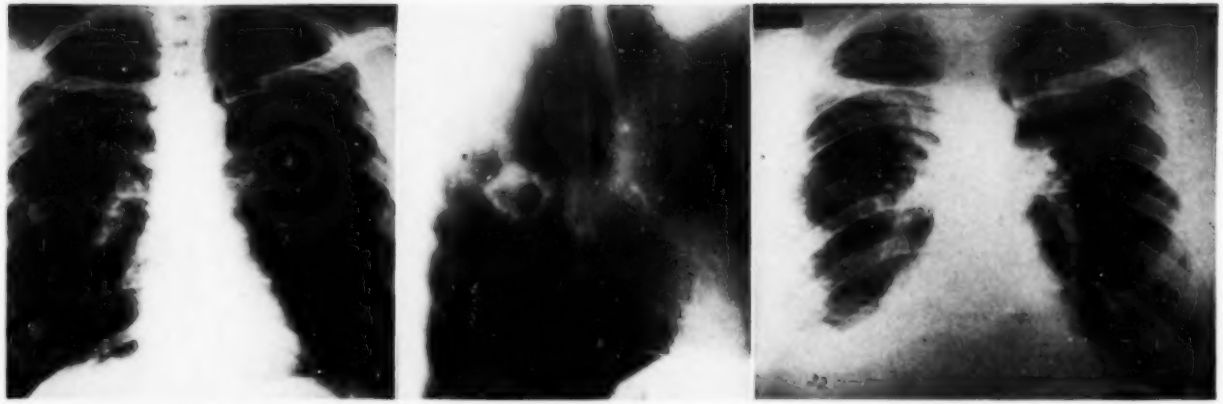

FIGURE 4A FIGURE 4B FIGURE 4C

Figure 4A: R.C., Male, aged 23 years, with a cavity in the superior segment of the right lower lobe, discovered in August 1950.—*Figure 4B:* R.C. The cavity is better localized in lateral tomogram.—*Figure 4C:* R.C. After five months' sanatorium treatment, the superior segment and the basal posterior segment which was found involved at the operation, were resected on February 2, 1951.

surgery. The cause of death was confiremd at autopsy. The second fatality occurred in a 25 year old woman on whom resection of the superior segment of the right lower lobe was easily accomplished. The patient's immediate postoperative condition was very good. About two hours after surgery a severe hemorrhage occurred. The chest was reopened, the bleeding segmental artery easily discovered and ligated, but the patient did not survive the second operation.

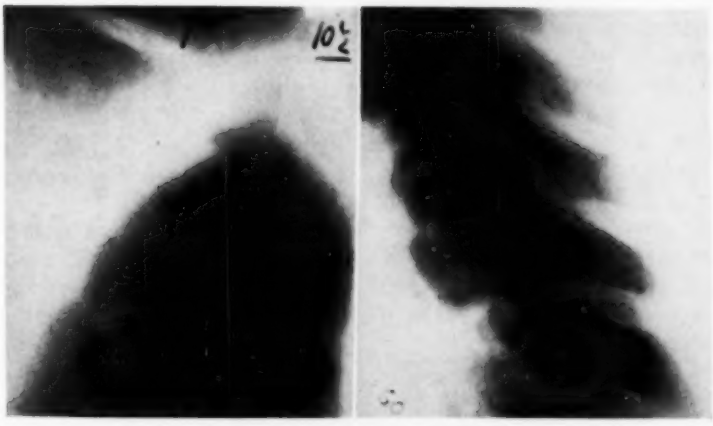

FIGURE 5A: A.R., Female, aged 27 years, treated since May 1950 by bilateral artificial pneumothorax for bilateral cavitary disease. On the right side two episodes of spontaneous pneumothorax with pericavitary infiltrates, increase of temperature and empyema occurred. The left lung cavity seemed to be under control (before the operation).

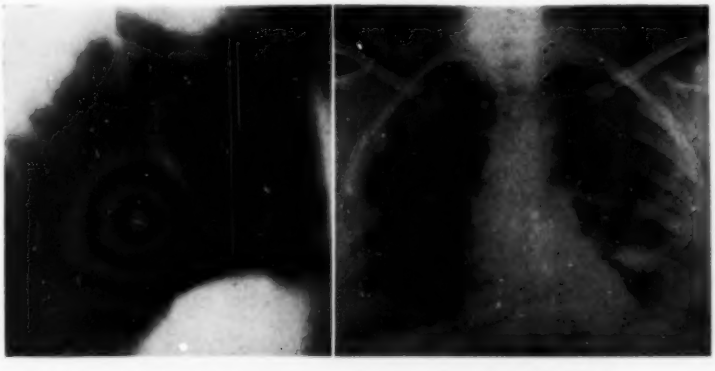

FIGURE 5B FIGURE 5C

Figure 5B: A.R. Lateral view of the right upper lobe cavity.—*Figure 5C:* A.R. Resection of the posterior segment of the right upper lobe and decortication of the lung has been performed on February 27, 1951.

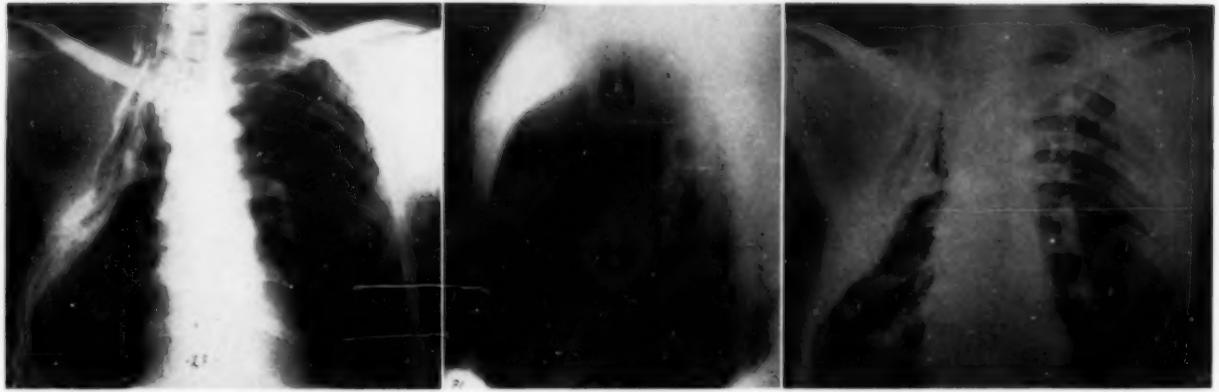

FIGURE 6A FIGURE 6B FIGURE 6C

M.J., Male, aged 28 years, had a small cavity in the left upper lobe, apparently controlled by artificial pneumothorax, when a right seven-rib thoracoplasty was performed for a large apical cavity on January 12, 1950. — *Figure 6A:* M.J. The left upper lobe cavity grew larger and pneumothorax was discontinued. — *Figure 6B:* M.J. Lateral view of the cavity. — *Figure 6C:* M.J. The posterior subsegment of the left upper lobe was resected on July 10, 1951.

Discussion

Collapse therapy, and particularly thoracoplasty, by its high incidence of sputum conversion and permanent closure of tuberculous cavities in well-selected cases, has demonstrated its value over a period of many years. There are, however, some forms of tuberculosis in which the ineffectiveness of collapse therapy in controlling the disease is generally recognized. This is true in cases of extensive unilateral tuberculosis or destroyed lung, endobronchial tuberculosis, lower-lobe disease, giant and tension cavities, tuberculosis with associated bronchiectasis, tuberculoma, mixed infection empyema with bronchopleural fistulae and uncontrolled pulmonary tuberculosis, and previous thoracoplasty or extrapleural pneumothorax failure. Lung resection offers such cases their only chance of cure and is very often considered a last resort procedure.

The main objection of the antagonists to pulmonary resection was, that tuberculosis is a generalized disease, and therefore one cannot hope to eradicate it by simple excision of one lung or a portion of a lung. In reality, tuberculosis is not necessarily a generalized disease in all its stages. Usually the tissue defences are able to deal with the greater part of the disease. The failure of the natural process of healing may result in cavitation in a quite localized area of the lung. Once cavitation has taken place the chances of spontaneous healing are diminished and spread to other parts of the lung is almost certain to occur (Bickford et al.[16]). It seems natural, then, that at any one time the most offending process may be confined to one or two segments of a lobe and the removal of the diseased area would avoid further spread of infection. The patient would thereby be given the chance to overcome lesser areas of infection by his natural defences supported by antibiotic therapy.

The refinements of the modern technique of segmental resection enables the surgeon to remove segments without causing any damage to the adjacent parenchyma and the low morbidity and mortality rates of this procedure may place this operation in the line of the safe surgical procedures. The most important factors are the proper selection of cases and choosing the optimum time for operation. The ideal indication for segmental resection is a localized disease confined to one segment of a lobe. The operation should be performed at an early stage of the disease before spread to other areas of the lung has occurred. We do not hesitate to treat by resection a patient presenting a localized disease who in the past had had a spread to the ipso- or contralateral lung, provided that the patient presents no clinical or radiological signs of activity in the preoperative period. In such a case segmental excision may be performed without great risk, and with a good chance of permanent cure.

There were, however, cases in which segmental resection was performed as a last resort procedure. These were considered as poor risks by the treating physicians, in whom all forms of conservative treatment have been tried to no avail and on whom collapse therapy was contraindicated. The surgeon, in general, is reluctant to such an approach, as these patients

have little chance of complete recovery after operations; on the other hand, any salvage must be considered as pure gain. These are patients with a tuberculous cavity in one segment, causing repeated spreads to other parts of the lungs. The removal of the source of seedings will put the organism in a better condition to control the more fresh exudative disease with the help of the available antibiotics.

The surgeon's very important task is to decide while the chest is open how much tissue to remove. He should determine by careful palpation of the whole lung the exact location and the limits of the main pathology. He will often find that the lesion is much more extensive than the radiological appearance would suggest. A large number of nodules of various sizes and consistency will be discovered in the healthy lobes or segments. One sometimes has great difficulties in judging about the degree of activity of small lesions. We as a rule leave behind small, hard, isolated nodules. On the other hand, we try to remove all the segments containing conglomerates of hard nodules or isolated soft nodules more than 0.5 cm. in diameter. If, however, a large number of elastic foci scattered throughout the lung are found at operation, without preoperative radiologic evidence, a most conservative excision is done. Resection is then limited to the segment containing the cavity, as it is quite possible that similar pathology exists in the contralateral radiologically clear lung. In the patients of Group III we always leave behind such foci, the decision being made before the operation.

Every patient is bronchoscoped as a routine prior to lung tissue resection. Whenever an endobronchial tuberculous disease of the main stem bronchus or of the lobar bronchi is discovered, the operation is postponed for fear of transecting and suturing of a sick bronchus. This may result in a bronchopleural fistula or persistence of a positive sputum for tubercle bacilli. It is, unfortunately, impossible to visualize bronchoscopically the segmental bronchi. It therefore quite often happens that the bronchus is diseased at the line of transection. In 11 cases, at least, of our series of 36 operations, some kind of tuberculosis at the line of transection was demonstrated by our pathologist. We believe that after the removal of the cavity which is the source of the infection, the endobronchial disease will clear up, particularly with the help of antibiotics. The postoperative course and the bronchial stump healing in these patients was as good as in others of the same group. One should not forget that also in the various types of collapse therapy the small sick bronchi are not removed; the incidence of healing and sputum conversion is nevertheless high.

Our immediate post-operative results seem to be gratifying. The incidence of complications is minimal. The most dangerous complication in excisional surgery is the bronchopleural fistula occurring in the early postoperative period and carrying a high mortality. We have had two cases of fistula (5 per cent) with non-tuberculous apical empyema; both healed promptly.

The mortality rate was, by and large, not higher than with lobectomy. One of the two fatalities is not directly related to lung excision or to surgery at all: This was a transfusion accident. The other was due to

postoperative hemorrhage. This was the only accident in a total of 225 resections, and should have a lesser frequency after segmental excision than after pneumonectomy, since one is dealing with much smaller vessels. No spreads or early exacerbations have occurred in our cases.

It is too early to judge final results as the first segmentectomy was performed only 16 months ago, and the majority of them within the last six months. We are unable to talk about the incidence of sputum conversion for the same reason, although follow-up results in the early period are very encouraging. In Chamberlain's series 86.7 per cent were culture negative.

As mentioned above, we did not accept for segmental resection those patients who, according to the experience in this country, may have been cured by means of a thoracoplasty or another form of permanent collapse therapy. At this early stage of our experience we did not want to compete with an orthodox established method of treatment. If, however, a three-year follow-up in a larger group of patients will prove the favorable early results, with a high incidence of sputum conversion in well-selected cases, we shall be inclined to perform limited resections in preference to thoracoplasty. Our decision is based on the following reasons:

Segmental resection is the most conservative procedure in the surgical treatment of pulmonary tuberculosis. The involved segment is usually smaller than a healthy one due to scarring, fibrosis, or atelectasis. Actually no lung tissue is sacrificed with the removal of such a segment and no respiratory function is lost. During the process of shrinkage of the segment the remainder of the lung parenchyma has undergone gradual distension. Not much more distension is required after removal of the sick segment, avoiding any strain upon a lesion which may have been left behind. Therefore no thoracoplasty has to be done after segmental excision.

Many years of experience have demonstrated that a good thoracoplasty may result in closure of cavities. It is nevertheless evident that the healing of a tuberculous cavity is a rather uncertain process and activation of a lesion apparently healed may eventually occur. By segmental resection the cavity is removed, and if there is no postoperative spread or exacerbation, the cure is more likely to be permanent.

Segmental resection, as compared with thoracoplasty, is a one-stage procedure with a much shorter period of postoperative hospitalization: on the average, 15 to 20 days in the uncomplicated case. The patients are more comfortable two days after that operation than after thoracoplasty and the convalescence is much smoother. This statement was volunteered by patients who underwent a segmental resection after a previous contralateral thoracoplasty. There is no deformity of the chest wall.

We think that the most important reason of preference of segmental resection to thoracoplasty is, that should, in the future, a new cavitary process develop in one of the lungs requiring surgical treatment, the surgeon will have a greater choice of procedures. Another excision, a thoracoplasty or extrapleural pneumothorax may be done without any fear, whereas after a good-sized thoracoplasty which necessarily resulted in limitation of the vital capacity, our choice would necessarily be limited.

Conclusions

Segmental resection constitutes the most conservative surgical treatment of pulmonary tuberculosis, and at present the only means of eradicating the disease at its early stage. It should be performed when after a period of six months treatment on accepted lines, including the use of antibiotics and chemotherapeutic agents, the disease becomes stationary and further healing is considered unlikely.

In well-selected cases, the immediate results are good, the morbidity minimal and the mortality rate low.

The operation should be considered as but one incident in the treatment, the patient being subsequently returned to the sanatorium and on appropriate regime.

SUMMARY

The place of segmental resection of the lung in the treatment of pulmonary tuberculosis is discussed.

The indications for the choice of this operation are reviewed, and a series of 36 cases presented.

The results in this series have been highly satisfactory, though final evolution must wait upon a longer follow-up.

RESUMEN

Se discute el lugar que ocupa la resección segmentaria en el tratamiento de la tuberculosis pulmonar.

Las indicaciones para la elección de esta operación se revisan y se presenta una serie de 36 casos.

Los resultados de esta serie han sido altamente satisfactorios aunque hay que esperar por mas largo tiempo de seguimiento de los enfermos para realizar una valuación final.

RESUME

La place de la résection segmentaire du poumon dans le traitement de la tuberculose pulmonaire est mise en discussion.

L'auteur examine les indications qui président au choix de cette opération et présente une série de 36 cas. Les résultats ont été très satisfaisants dans ces séries, bien qu'on ne puisse parler d'évolution complète avant d'avoir suivi le malade pendant beaucoup plus longtemps.

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Myocardial Involvement in Generalized Scleroderma*

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Introduction

Generalized scleroderma is no longer considered strictly a dermatosis, but rather a disease of the connective tissue of various organs and systems. The pathological changes characterizing scleroderma have been thoroughly described and reviewed by Beerman.[4] Of recent years, numerous investigators[1,3,4,6-7,9,12,14,16-17,19-22] have emphasized the frequency of myocardial and pericardial involvement in generalized scleroderma. In fact, Weiss et al.[22] pointed out that in their series of nine cases, three presented cardiovascular symptoms several years prior to the development of skin lesions and concluded that scleroderma heart disease is a clinical and pathological entity. Consequently, both the internist and dermatologist must be aware of the possibility of cardiovascular involvement in this disease. The purpose of this communication is to present an additional case of scleroderma heart disease.

Case Report

First Admission:

L. J., a 50 year old colored male, entered the Cardiovascular Section of Kennedy Veterans Administration Hospital, March 8, 1947, with a chief complaint of shortness of breath. The onset of his present illness dated back one year when he noted exertional dyspnea which increased in severity so that at the time of admission he was having definite orthopnea. He also complained of soreness beneath the anterior chest wall and pain in the right axilla. He was conscious of constant palpitation. For approximately a year he had noted a productive cough which had become more severe in the two months preceding admission. There was no history of hemoptysis or dependant edema. The history by systems revealed only that he had a penile lesion prior to World War I. The past medical, social, and family history was otherwise non-contributory.

Physical Examination: The patient was a well developed and well nourished negro male with evident dyspnea. The pertinent findings were limited to the cardiovascular system. The heart sounds at the base were faint and tachycardia was present. The blood pressure was 140/88. There were inconstant and scattered musical rales over the entire posterior chest but most prominently at the bases where they were more pronounced on deep expiration. The liver was slightly enlarged.

Laboratory Studies: Serological tests for syphilis were positive. A blood count was normal except for a differential revealing 65 per cent lymphocytes, and a heterophile test was positive 1:28. Electrocardiographic studies (Figure 1) showed elevation of the ST segments over the right side of the heart.

Clinical Course: He was seen by the cardiac consultant who felt that all symptoms were adequately explained by a diagnosis of bronchial asthma. He was treated with aminophyllin and adrenalin and improved. He was discharged with diagnoses of bronchial asthma and latent syphilis.

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Second Admission:

He was readmitted to the cardiovascular section, February 6, 1948. He had done fairly well until two months prior to admission when he noticed swelling of his feet and ankles toward evening. This was followed by swelling of the arms and face. He also complained of fever, soreness in his arms, legs and most of his joints. His fever subsided but the joint and muscle pains continued up to admission. Attacks of dyspnea persisted.

Physical Examination: The patient was still well developed and nourished. Fundoscopy revealed slight tortuosity and narrowing of the retinal vessels. The ability to open his mouth was restricted to 60 per cent of normal, and he stated that this had been present for several weeks. The percussion note over the chest was hyperresonant. Asthmatic wheezes were audible at both bases and there were decreased breath sounds at the apices. The blood pressure was 115/76. A^2 was equal to P^2 which was accentuated. The cardiac sounds were of good quality and occasional extrasystoles were present. The liver was palpable one fingerbreadth below the costal margin. There was no edema of the legs, arms or face. The joints were clinically normal.

Laboratory Tests: The urinalysis was negative and a Fishberg test revealed a

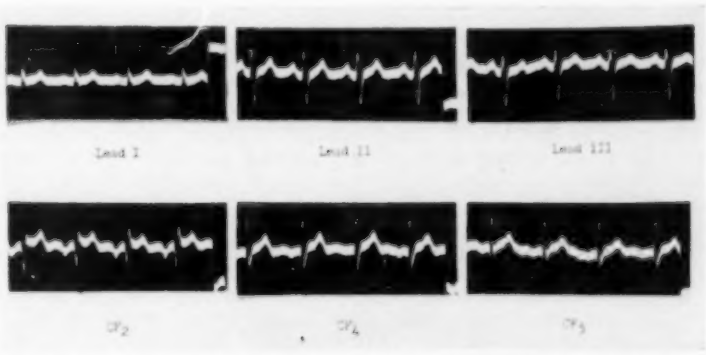

FIGURE 1: This electrocardiogram was taken on the patient's first admission. The ST takeoff in CF^2 is somewhat elevated. Otherwise the tracing is probably within normal limits.

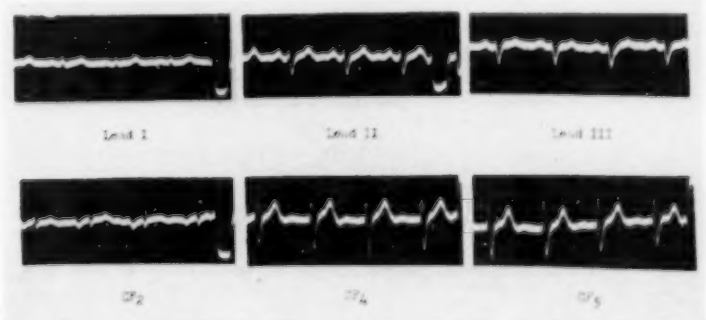

FIGURE 2: This tracing was taken approximately 11 months following figure 1. There has been a marked lowering of the voltage in the three standard leads and CF^2. The transition point has now moved from CF^4 to CF^5.

concentration of 1.022. The serological tests for syphilis were again positive. The blood count and hemoglobin were normal, but the differential revealed 44 per cent neutrophils, 48 per cent lymphocytes and 8 per cent eosinophils. The sedimentation rate (Westergren) was 23 mm. per hour. X-ray film and fluoroscopy of the lungs and heart were normal except for a suggestion of prominence of the pulmonary conus in the right anterior oblique position. Joint x-ray films were normal but minimal calcifications were present in the arterial walls in the legs.

Clinical Course: While in the hospital he ran a low grade fever. Symptomatic treatment for the asthmatic state gave satisfactory relief. An electrocardiogram taken February 6, 1948, showed a low voltage and clockwise rotation (Figure 2). A second tracing taken February 25, 1948, however, showed the sudden appearance of incomplete right bundle branch block (Figure 3). At this time it was felt there was enough evidence to make diagnoses of acute rheumatoid arthritis and cor pulmonale. He was discharged following therapy and was instructed to report to his local health department for treatment of latent syphilis.

Final Admission:

He was readmitted on June 6, 1948, with progression of joint and muscle pains. For two weeks preceding this admission, he was unable to walk or attend to his personal toilet.

Physical Examination: He appeared chronically ill, was lucid, oriented, and cooperative, but had difficulty in opening his mouth. There was wasting of the shoulder girdle muscles. The chest was hyperresonant to percussion and asthmatic wheezes were heard over both lung fields. Examination of the heart revealed weak and distant sounds. The rate and rhythm were normal. P^2 was louder than A^2. The blood pressure was 118/80. The remainder of the examination was negative except for moderate tenderness of the muscles and joints of both the upper and lower extremities.

Laboratory Data: The blood count was within normal limits. The sedimentation rate was 24 mm. per hour. Urinalysis was not remarkable. NPN was 23 mgm. per cent. The total protein was 6 grams with an A/G ratio of 1. Serological tests for

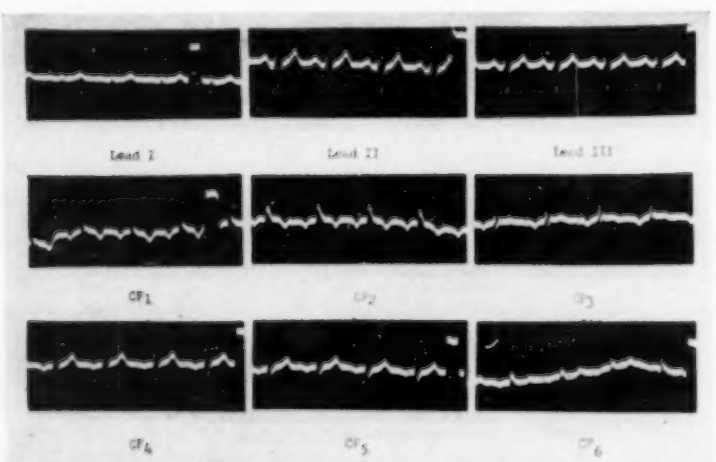

FIGURE 3: This electrocardiogram was taken approximately two and a half weeks following the tracing in figure 2. The three standard leads do not reveal any gross change. However, there is a marked change in the CF leads. Incomplete right bundle branch block is now present.

syphilis were repeatedly positive in low titer. The spinal fluid examination was normal. Liver function tests, blood electrolytes and urinary 17 ketosteroids were within normal limits. The sputum was negative for acid-fast organisms. Chest x-ray films showed evidence of only pulmonary emphysema, and a barium enema was negative. KUB films revealed only calcifications of pelvic vessels. An esophagram demonstrated a dilated esophagus with markedly diminished peristalsis. Joint x-ray films again were negative except for minimal osteoarthritis of the spine. An electrocardiogram taken June 10, 1948, showed a disappearance of the right incomplete bundle block seen February 25, 1948, and in addition showed a supraventricular premature systole with aberrant conduction (Figure 4). A final tracing taken October 14, 1948, showed further lowering of the voltage with more frequent supraventricular premature systoles in addition to an increase in the duration of the QRS interval to 0.12 seconds (Figure 5).

Clinical Course: On admission this was felt to be a case of rheumatoid arthritis or possibly fibrositis. However, it was soon noted that the skin was becoming shiny and inelastic and that the facial expression was masklike. Generalized scleroderma was suspected at this time. On palpation of the arms and legs there was a distinct leathery feel of the skin and subcutaneous tissues. It was at this time that the esophagram revealed the dilated esophagus with only slight peristalsis. However, chest x-ray films revealed no definite heart or lung changes. A biopsy of the skin from the anterior abdominal wall was reported as consistant with generalized scleroderma. He ran a slowly progressive downhill course with occasional elevations of temperature to 101 degrees F. An extensive course of penicillin given for his latent syphilis did not affect his failing health. Physiotherapy afforded only slight symptomatic relief. During the last month of hospitalization, severe dysphagia developed with frequent regurgitation of food which was often blood

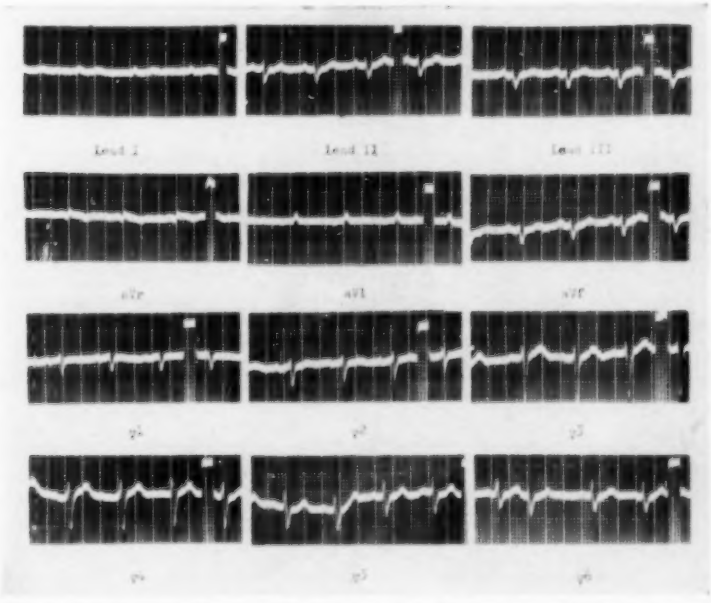

FIGURE 4: The incomplete bundle branch block seen in figure 3 is no longer present. The voltage has decreased even further. The transition point is now present in V^6. A supraventricular premature beat with aberrant conduction is seen in V^6.

stained. He became absolutely helpless, being unable to raise his hands from the bed. He continued in this state of inanition and quietly expired on October 27, 1948.

Necropsy: Examination of the body disclosed loss of much subcutaneous fat and a thin, smooth, shiny skin which had lost its elasticity. The skin was stretched over the bony prominences and showed cracking and scaling in some areas. There was generalized muscle wasting; the pectorals were thin, atrophic and pale. Over the sacrum was a large decubitus measuring 6 cm. in diameter.

Each pleural cavity contained 200 cc. of clear yellow fluid. There were a few fibrinous interlobar adhesions. The pericardial sac contained 100 cc. of a similar fluid. Section of the lungs revealed moderate congestive changes and a few emphysematous blebs.

The heart weighed 180 grams and showed edema of the subepicardial fat. In the left and right ventricular walls were depressed, scarred areas measuring up to 1 x 0.3 cm. The valve measurements were normal, but along the edges of the mitral and tricuspid valves were healed verrucous lesions. The myocardium was firm, and the scars noted on the surface extended in some areas through the entire thickness of the wall. The uninvolved left ventricular wall measured 1.2 cm.; the right 0.4 cm. The coronary vessels were moderately sclerotic and tortuous but their lumina were patent.

The kidneys each weighed 100 grams. The capsules stripped with difficulty. The cortico-medullary demarcation was indistinct and the cortex measured approximately 0.5 cm. The calyces, pelves and ureters were not remarkable.

The esophageal mucosa showed numerous irregular plaque-like whitish elevations and areas of ulceration. There was moderate thickening and firmness in the esophageal wall.

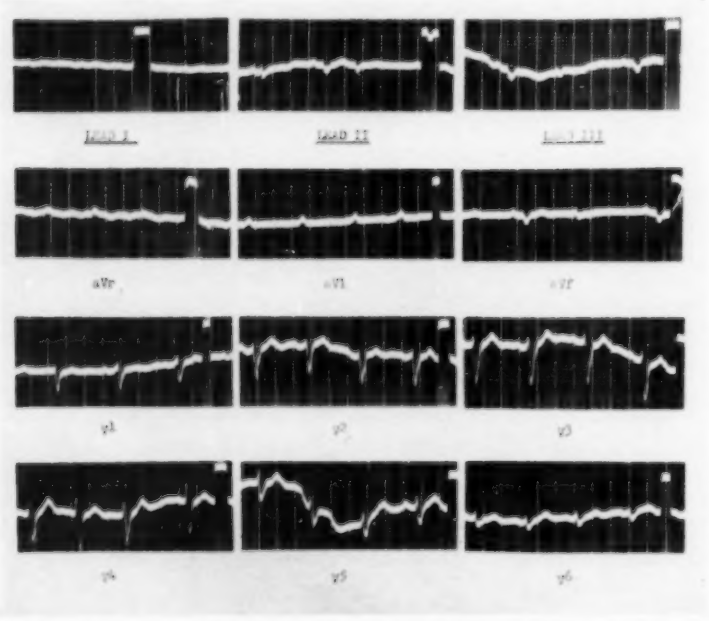

FIGURE 5: This tracing was taken four months following figure 4. The voltage in the standard leads continues to decrease. A supraventricular premature beat is present in Lead 2 and Lead 3. A nodal premature beat is seen in aVf. The S waves in Lead 3, aVf and V^5 and V^6 are now greatly slurred.

The remainder of the necropsy revealed only moderate testicular atrophy and gynecomastia.

Microscopic Study: The sections from the skin disclosed atrophy of the epidermis and an increase in the number of collagen fibers in the dermis. These fibers were thick, dense, often fragmented, and stained deeply with eosin. Skin appendages were decreased in number and atrophic. Small collections of fat cells were caught in the dense scar and showed an embryonic appearance. Small vessels were concentrically thickened.

In the lungs there was an increase in collagenous material in the interalveolar septa and peribronchial scarring. The small branches of the pulmonary arteries showed moderate to marked concentric thickening and decrease in size of the lumen.

Sections from the heart disclosed moderate epicardial fibrosis which contained flecks of calcified material and a few round cells. Similar calcifications have been noted by Durham[5] (Figure 6). Throughout the myocardium there was a general increase in connective tissue, varying from slight interstitial cellular fibrosis (Figure 7) to large, dense, hyaline scars (Figure 8). Frequently the large areas of scarring were hyalinized in the center and more cellular at the periphery where the connective tissue strands interlaced with and often imprisoned small groups of myocardial fibers. These trapped fibers exhibited fracturation, vacuolization of cytoplasm, and other degenerative changes. Nearby were scattered hemosiderin-laden macrophages and occasional chronic inflammatory cells. The distribution of the scarring was diffuse and had no constant relation to the coronary vascular tree. Often the areas of fibrosis contained numerous small patent vessels (Figure 8). The small arteries demonstrated both intimal and medial concentric thickening to a marked degree, and the capillaries had a prominent endothelial lining. The muscle fibers in the uninvolved areas of the myocardium showed no significant changes.

In the kidney sections stained with hemotoxylin and eosin there was noted only slight increase in intercapillary hyaline material, but the true extent of this deposition was brought out by the periodic-acid Schiff stain. Only occasional intralobular arteries revealed concentric hypertrophy, and afferent arterioles with thickening or fibrinoid necrosis were rare.

The esophageal lining was ulcerated in many areas. The underlying wall was thickened by dense fibrosis which involved the muscle layers.

Sections from striated muscle showed changes somewhat similar to the lesions in the heart. Many of the fibers were atrophic or degenerating. Many had lost their striations and were hyalinized and fracturated. Dense bands of hyaline and cellular connective tissue coursed through the sections and scattered collections of lymphocytes were present.

Discussion

Brock[3] in 1934 reported a case of generalized scleroderma in which the cause of death was congestive failure due to myocardial fibrosis. In 1943 Weiss et al. summarized the findings in nine cases of generalized scleroderma in three of which cardiac symptoms preceded the skin changes by two years. Goetz et al.[8-10] in 1942, 1945 and 1951 emphasized the frequency of myocardial changes in this disease. In recent years Mathisen and Palmer,[14] East and Oram,[6] Spain and Thomas[19] and Ries[17] have contributed additional studies on scleroderma heart disease. Several investigators[2,13] have reported pericarditis occurring in scleroderma heart disease. Pericardial fibrosis was prominent in the patient presented in this case report.

It is interesting that this case presented cardiovascular symptoms ap-

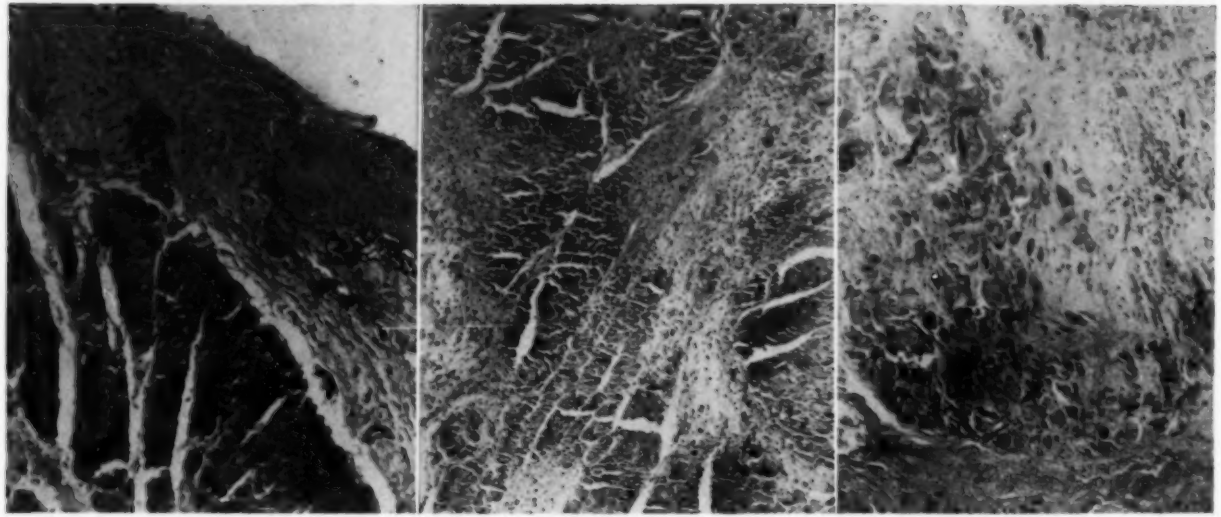

FIGURE 6 FIGURE 7 FIGURE 8

Figure 6: Heart. Right Auricle showing fibrous thickening and areas of calcification in epicardial region. (H & E x 100).—*Figure 7:* Heart. Left Ventricle showing cellular and hyalin fibrosis. Many groups of muscle fibers are being surrounded by the cellular fibrous tissue (H & E x 100).—*Figure 8:* Heart. Left Ventricle showing fibrosis. Note patent small blood vessels and trapped degenerating myocardial fibers. (H & E x 200).

proximately a year before the actual diagnosis became apparent as in three of the cases of Weiss et al.,[22] and the case of Spain and Thomas.[19] Increasing dyspnea is the most common presenting symptom in scleroderma heart disease, being present in every case reported in the literature. This is probably due mainly to the progressive myocardial fibrosis and partly to the changes in the lungs. Chest pain, such as experienced by the patient, is a frequent occurrance.[6,14,22] No explanation has been given for its origin, though it probably is the result of the pathological changes in the myocardium or pericardium. During the progress of the disease, P^2 increased in intensity. This has been emphasized by Weiss et al[22] in their series.

Numerous changes have been reported in the electrocardiogram in scleroderma heart disease[1,7,12,14,16,22] The most frequent finding is low voltage. Arrhythmias such as auricular or ventricular premature systoles, auricular fibrillation, various degrees of AV block, and bundle branch block have been noted. This patient presented low voltage, supraventricular premature systoles, and a transient episode of right bundle branch block. Incomplete bundle branch block was present in three of Gil's seven cases.[7]

Both the gross and microscopic pathological findings were typical of those described in generalized scleroderma.[4] The changes were most pronounced in the heart, esophagus, skin and muscle, but a generalized increase in connective tissue was present also in the lungs and kidneys.

In the heart the pathological changes showed different stages of development. These changes were not found in the distribution of any one large coronary vessel such as may follow occlusion of an artery. The changes were diffuse throughout the myocardium indicating generalized involvement of focal areas and smaller vessels. These vessels showed marked concentric thickening and were of the small artery size or smaller. The sequence of the changes in the heart consist first of focal edematous cellular and vascular fibrosis. This gradually becomes more compact and finally resolves into a dense hyaline scar. Often the process remains active about the periphery of the involved areas and myocardial fibers are trapped and degenerate and disappear. Whether the vascular changes precede the focal scarring is not known, but possibly the same underlying mechanism is active in both the vessels and myocardium at the same time. Primary vascular involvement is suggested by the fact that numerous patients with scleroderma have previously exhibited Raynaud's phenomenon. No inflammatory changes in the arteries or arterioles were observed in this case. Though the etiology of scleroderma remains obscure, the changes in the heart present a distinct pathological picture. There was no granulomatous inflammation, gummatous changes, or marked inflammatory reaction to suggest luetic myocarditis,[18] and the aorta demonstrated none of the changes of syphilis. The findings of progressive lowering of blood pressure and low voltage in the ECG tracings are easily explained by the progressive diffuse scarring in the hearts of patients with scleroderma. The various other ECG changes must follow involvement of specific areas.

This patient had no significant urinary findings during life and yet

revealed kidney lesions to a limited degree, similar to those described by Moore and Sheehan.[15]

SUMMARY

A case of diffuse scleroderma (progressive systemic sclerosis) with autopsy is presented and discussed. Symptoms of heart disease were the first to appear and were followed by involvement of joints, muscles, skin and esophagus. The related literature is reviewed.

RESUMEN

Se presenta y se discute un caso de escleroderma difuso (esclerosis general progresiva). Los síntomas cardiacos fueron los primeros en aparecer y fueron seguidos de afección de las articularciones de los músculos de la piel y del esófago. La literatura al respecto se revisa.

RESUME

L'auteur rapporte et discute une observation de sclérodermie diffuse avec autopsie. Les premiers symptômes furent des troubles cardiaques que suivirent une atteinte des articulations, des muscles, de la peau et de l'oesophage. Cette observation est suivie d'une revue de la littérature.

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Teaching Cardiology to Undergraduate Students

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Modern cardiology is based on knowledge derived from several disciplines: Anatomy and embryology, physiology, pathology and bacteriology, pharmacology, physics, roentgenology, physical diagnosis, comparative biology, general medicine and surgery, pediatrics, and therapeutics. Psychiatry in all its aspects, public health, industrial organization, and social service, have multiple problems in common with cardiology. This strict connection between cardiology and allied or parent sciences is demonstrated by a few examples: Conduction disturbances (anatomy); complex malformations of the heart (embryology); rheumatic fever (bacteriology, immunology, preventive medicine); neurocirculatory asthenia (psychiatry).

Teaching of cardiology cannot be entirely separated from any of these various contributory disciplines and it could be said that cardiology cuts transversely through all the above mentioned fields.

Teaching of several aspects of cardiology can be done in the respective departments following agreements between the program director and the various chairmen. This applies to anatomy, embryology, bacteriology and pathology, biology, psychiatry, physics, physical diagnosis, and surgery.

Certain basic data of cardiology can still be taught in the respective departments but should be integrated by additional lectures given by the program director or his co-workers. This applies chiefly to physiology, pharmacology, roentgenology, public health, and pediatrics. In our school, two lectures of physiology (the electrocardiogram, the heart sounds); two of pharmacology (digitalis bodies; quinidine and other drugs); two of pediatrics (congenital heart diseases); and one of public health (prevention of cardiovascular diseases) are given by the program director within the frameworks of other courses.

Other aspects of cardiology, which are usually part of the course of medicine, are so basic that they should be taught entirely by the teachers of cardiology. Twelve lectures of didactic medicine dealing with the various cardiovascular diseases, 12 lectures of elementary electrocardiology, 24 cardiac seminars, 24 cardiac clinics, and two pediatric seminars, serve this purpose. A 12-lecture elective course of *advanced cardiology* completes the specific knowledge of the students.

Additional clinical lectures are given by various instructors of medicine, and clinical pathological conferences are given by instructors of pathology, according to the clinical material.

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The fourth in a series of articles prepared under the sponsorship of the Council on Undergraduate Medical Education, American College of Chest Physicians.

The general structure of cardiology teaching in shown in Table I.

TABLE I: LECTURES GIVEN BY CARDIOLOGY STAFF

	Introductory	*Theoretical*	*Practical or Clinical*
First year	2-seminars 2-physiology		2-physiology (lab.)
Second year	2-pharmacology	12-didactic medicine 2-didactic pediatrics	2-pharmacology (lab.)
Third year		1-public health**	12-electrocardiography** 12-cardiology (clinical cases)** 2-4 CPC* ** 2-pediatrics (seminars)**
Fourth year		12-advanced cardiology**	24-cardiology** (seminars) 24-cardiology** (clinics) 2-4 CPC* **

*Staff takes part in discussion.
**Given two or more times to groups of students.

Various trends in the teaching of cardiology are revealed by the way it is considered by the Academic Staff and the school authorities.

(a) *Cardiology is part of medicine.* There is no need for separate teachers or specialized courses. The latter would tend to create specialists instead of general practitioners.

(b) *Cardiology is part of the teaching of chest disease* within or without the courses of medicine. The various diseases of the heart, lungs, mediastinum, and vessels, are taught by the same instructors.

(c) *Cardiology is a subdivision of medicine which should be taught within the course of medicine* by different teachers. Cardiology teaching is supported by a *division of cardiology* which is connected with the teaching hospital and is a section of the department of medicine of the school.

(d) *Cardiology is a separate discipline* which cuts across the others. It should be taught by special teachers, in a *separate department of cardiology.*

The first statement corresponds to a viewpoint which was correct at the beginning of the century but which is definitely obsolete now. The second statement couples two different kinds of specialties (heart and lungs) which, even if they have multiple points in common, should be kept separate from a didactic and practical point of view.

Cardiology *is part of* physiology, pharmacology, pathology, or medicine according to the emphasis given to some of its aspects for elementary teaching. It is *neither of them* as soon as its more advanced aspects are reached, like electrocardiology or its refinements, other graphic methods including phonocardiography, electrokymography and ballistocardiography, catheterization of the heart, and angiocardiography. These technical branches and, most important, their correlation with the clinical data, can be presented by specialists only. These specialists illustrate the most

elementary aspects to undergraduate students while the highest refinements should be left for postgraduate teaching.

Either of the trends listed as (c) and (d) can be accepted according to the conditions of the school; these include financial support, availability of trained and skilled personnel, possibility of overcoming older traditions and personal jealousies, and general progressive trend of the school authorities. Either of them can be made to work.* It should not be forgotten that, while certain refinements of cardiology require discussion by specialists, others can and should be presented by internists or even by practitioners. Thus, statement (a) can be reconciled with statement (c) if both aspects of teaching are developed and presented, side by side, to the student body.

It is the opinion of the writer that a separate department of cardiology, working in close collaboration with the departments of physiology, roentgenology, and medicine, is the best solution. Such a department should include:

(a) experimental section and animal surgery;
(b) laboratory of clinical electrocardiology;
(c) laboratory of graphic studies;
(d) laboratory of catheterization and angiocardiography;
(e) clinical ward;
(f) out-patient department (cardiac clinics).

It is likely that a perfect solution can be found only in schools having a teaching hospital where teaching comes first and cannot be subordinated to other considerations.

Conclusions

Cardiology should be considered from three separate points of view:

(a) As part of various basic sciences or other independent disciplines.
(b) As part of medicine.
(c) As a specialty in itself.

All three aspects should be taken into consideration in the planning of undergraduate teaching because omission of any of them would lead to incomplete, obsolete, or specialized knowledge.

*In our School, trend (c) is now followed, while chairs of Cardiology have been recently established in Georgia.

Editorial

THE PROBLEM OF VISCERAL LUNG FUNCTION

The need for and justification of a proposed concept of lung function is discussed in a review article featured in this issue. The author in a frank partial statement of his concept of visceral function of the lungs proposed by him almost two decades ago is also frank in pointing out its wholly theoretical nature. This he endeavors to justify by considerations which we seriously believe deserve a hearing.

Perhaps many readers will question the wisdom of presenting a concept so speculative in nature. However the present confused state of our knowledge of pulmonary function and dysfunction particularly justifies any new approach, even other than those currently used. It is true that modern ingenious techniques (catheterization and angiography of the pulmonary vessels) recently added to older proved methods of spirometric and gas analytical studies have vastly increased our knowledge in this field. But, it is also true that recent observations have revealed a number of puzzling problems, for which no explanation exists except in theory. The lungs are perhaps the most inaccessible organ to direct exploration and observation and we must realize the need of much more investigation. With this, great opportunity for speculation still exists in order to fathom the intricate nature of the function of such inacessible ultimate pulmonary structures.

Perhaps most of us will therefore agree that the writer has shown fairly the inadequacy of the mechanistic concepts now prevailing. He has further shown the need for consideration of the concept of visceral lung function as a working theory that can logically afford an answer to some of the unsolved problems of pulmonary physiology.

The concept has a strong appeal to our biological thinking. It fits in well with the general scheme of other visceral functions of the body. Even though still inferential in nature, it is supported by some substantial evidence indicating that the ultimate structures of the lungs undergo the changes assumed in their hypothetical function and that these structures function in a manner revealing some regulation possibly by a specific hormonal action.

Last but not least the value of this concept lies in its clinical application. The plausible explanation it affords of the nature of pulmonary disease related to emphysema has particularly stimulated our interest. It has led us to believe in a common denominator in emphysema. Our oncept of "air space disorder" is that of a pathological manifestation associated with disturbed visceral function of the lungs. As pointed out elsewhere* this concept appears to us a logical answer to the problem of emphysema which has so long remained unexplained.

Should this concept fail to be confirmed, the author will have served at

*Mayer, Edgar and Rappaport, Israel: "Clinical Observations in Airspace Disorder of the Lungs. A New Concept," *J.A.M.A.*, in press.

least a useful purpose in calling attention to a general lack of awareness of the existence of a most fundamental aspect of pulmonary physiology, namely, the problem of the true nature of lung function. This article deserves careful study and appreciation for the much needed stimulation it affords to a new approach to the problems of lung function and dysfunction. It will no doubt afford incentive for much thinking and research along these lines. The idea indeed is so timely that work is already under way toward unraveling the secret of visceral lung function. However simple the concept itself is, the necessary research is most intricate; and much difficult work will be required for its solution.

Edgar Mayer.

Semi-Annual Meeting, Board of Regents

The Semi-Annual Meeting of the Board of Regents, American College of Chest Physicians, was held at the Sheraton Hotel, St. Louis, Missouri, on Monday, November 30, 1954. The following Regents, Governors, council and committee chairmen and guests were present:

Donald R. McKay, Buffalo, New York, Chairman
Russell S. Anderson, Erie, Pennsylvania
Albert H. Andrews, Jr., Chicago, Illinois
Andrew L. Banyai, Milwaukee, Wisconsin
Otto L. Bettag, Chicago, Illinois
Charles A. Brasher, Mt. Vernon, Missouri
Edward Dunner, St. Louis, Missouri
Seymour M. Farber, San Francisco, California
Carl H. Gellenthien, Valmora, New Mexico
Alfred Goldman, St. Louis, Missouri
Alfred Goldman, Los Angeles, California
Burgess L. Gordon, Philadelphia, Pennsylvania
Alvis E. Greer, Houston, Texas
William A. Hudson, Detroit, Michigan
Hollis E. Johnson, Nashville, Tennessee
Harold I. Kinsey, Toronto, Ontario
Louis Mark, Columbus, Ohio
Elliott Mendenhall, Dallas, Texas
Jay Arthur Myers, Minneapolis, Minnesota
J. Winthrop Peabody, Washington, D. C.
Charles K. Petter, Waukegan, Illinois
Joseph C. Placak, Cleveland, Ohio
A. J. Steiner, St. Louis, Missouri
James H. Stygall, Indianapolis, Indiana
Harold G. Trimble, Oakland, California
David H. Waterman, Knoxville, Tennessee
Roy A. Wolford, Washington, D. C.
Murray Kornfeld, Executive Director, Chicago, Illinois
Harriet L. Kruse, Executive Assistant, Chicago, Illinois

Dr. McKay called the meeting to order at 2:00 p. m. and requested that the report of the Treasurer be read. Dr. Petter presented the financial statement of the College to the present date and the proposed budget for 1954. He further reported that the Committee to Study the Group Insurance Plan of the College, of which the President had appointed him chairman, had begun their investigation and hoped to be able to submit recommendations in the near future. The report of the Treasurer was unanimously adopted in full, upon motion duly made and seconded.

Dr. Gellenthien, chairman of the Committee on By-Laws, presented the following recommendations:

> It is recommended that the College By-Laws be amended to read that all applicants for membership be admitted to either Associate Fellowship or Associate Membership, with the exception of those candidates who are admitted directly to full Fellowship without examination. (Article III, Section 7, page viii.)
>
> It is recommended that the office of Assistant Treasurer be added to the list of officers which is presented annually by the Committee on Nominations. (Article X, Section 5, page xvi.)
>
> It is recommended that the Chairman of the Council on International Affairs be appointed by the President to serve for a period of two years, as stated in the By-Laws, and that the remainder of that sentence, i.e., "and is to be from the country where the International Congress on Diseases of the Chest is to be held" be removed. (Article XII, Section 5, page xix.)

Upon motion duly made and seconded the recommendations of the Committee on College By-Laws were adopted and will be voted upon by the College Membership at the next annual Administrative Session to be held in San Francisco, California, on Saturday, June 19, 1954.

Dr. Farber, chairman of the Committee on General Arrangements for the 20th Annual Meeting of the College to be held in San Francisco, June 17-20, 1954, reported that work had begun on the setting up of local committees which would be directly responsible for the arrangements and function of the various activities.

Dr. Anderson presented the report of the Council on Hospitals and its subcommittees. The Board of Regents was asked to give official approval of the minimum salary recommendations prepared by the Committee on Standards and Accreditation and published in the September, 1953 issue of *The Hospital Counselor*. Considerable discussion followed and upon motion made and seconded, the matter was referred back to committee for further study.

Dr. Bettag reported briefly on the activities of the Committee on Chest Diseases in Institutions and made particular reference to the recent establishment of tuberculosis-neuropsychiatric hospitals, one of which was opened on November 13 at the Veterans Administration Hospital, Downey, Illinois. Dr. Wolford pointed out that this was only one of thirteen hospitals of this type to be established by the Veterans Administration.

Dr. Edward W. Hayes, Monrovia, California, was not able to be present and as chairman of the Council on Undergraduate Medical Education his report had been prepared in copy form and was brought to the attention of the Board. Dr. Trimble reported that he had accepted the responsibility, upon the request of Dr. Hayes, to assist in the collection of teaching material in the form of x-ray pictures, slides and short films which may be duplicated and made available to all medical schools. Two other activities of the Council were reported, namely, that a series of short articles on the various phases of teaching diseases of the chest were being prepared by Dr. Theodore H. Noehren, Vice-Chairman of the Council, for publication in *Diseases of the Chest*, and that the Council was working in cooperation with the Committee on College Essay Award in promoting the prize essay contest which at the annual meeting of the Board of Regents had been voted to be limited to medical students.

Dr. Banyai, chairman of the committee for the book on "Non-Tuberculous Diseases of the Chest" reported that the index for the book was almost finished and that the book would be published in the near future.

Dr. Bettag, reporting on the activities of the Committee on Chest X-ray, which is a joint committee with the American College of Radiology, stated that the two societies were coordinating their efforts in having the report of the joint committee published in the state and local medical journals.

Dr. Peabody, chairman of the Council on Postgraduate Medical Education, requested that the assembled members stand for a moment in silent tribute to Dr. Frank R. Ferlaino, recently deceased, who had served faithfully as Secretary of the council. Dr. Peabody then reported on the postgraduate course program of his council. (A report of recent and coming postgraduate courses was published in the November, 1953 issue of *Diseases of the Chest*.)

Dr. Hudson, in reporting for the Board of Examiners, of which he is chairman, recommended that the Fellowship examinations be held at the semi-annual meeting as well as at the annual meetings of the College in order that the burden of examining and grading may be divided. He stated that this would afford an opportunity for the oral examiners to devote more time to each candidate. It is the feeling of the Board of Examiners that insufficient time has been devoted to the oral examinations and the members of the Board are of the opinion that the additional time would give them a clearer perception of the candidates' qualifications. Dr. Hudson further recommended that candidates for Fellowship

in the College be screened more thoroughly at the ground level and that each candidate be required to have at least two sponsors. Upon motion duly moved and seconded, the recommendation that Fellowship examinations be held twice yearly, at the annual and semi-annual meetings of the College, was unanimously adopted. The second recommendation of the Board of Examiners was referred to the committee to study membership requirements.

In the absence of Dr. H. J. Moersch, Rochester, Minnesota, the chairman of the Council on Research, the members of the Board of Regents were referred to mimeographed copies of his report which had been distributed. The report covered the activities of the various committees serving under the Council on Research. Dr. Trimble reported personally on the activities of the Committee on Non-Surgical Collapse Therapy and announced that the statistical work on the study of the ten cases of pneumothorax is now being completed. He further reported that the study on pneumoperitoneum has reached the point where statistical data has been collected, the code for the punch cards is worked out, and that the information will shortly be compiled.

Dr. Alfred Goldman, Los Angeles, reported on the activities of the Committee on Surgical Treatment of Diseases of the Chest. The committee has made a survey of carcinoma of the lung and follow-up of surgical treatment and now has data on well over 150 cases, which Dr. Goldman stated, were very encouraging. It is planned to continue the study for several more years and follow-up on several hundred cases for five years.

Dr. Andrews, secretary of the Committee on Physiologic Treatment, presented a report of progress on procedure approved by the Board of Regents at their annual meeting in May, 1953:

1) A course for inhalational therapy technicians was given at the Alexian Brothers Hospital, Chicago, November, 1953. Attendance was greater than at previous courses.
2) Plans for the postgraduate course on inhalational therapy are proceeding but are not sufficiently advanced to permit a definite report at this time.
3) The research of Dr. Andrews on continuously recorded oxygen analysis, endorsed at the past meeting of the Board of Regents, has progressed. A grant of $21,000 has just been received at St. Lukes Hospital, Chicago, to support this study.
4) The exhibit on inhalational therapy is nearly completed and awaits the procurement of funds.
5) Conferences with the American Medical Association and the American College of Surgeons have resulted in progress on the development of standards and certification for inhalational therapy technicians. At a recent conference with officials of the American Hospital Association, the need for standards of inhalational therapy for hospitals was recognized.

Dr. Andrews then presented the following motion which had been approved by the Executive Council at their meeting in St. Louis on November 29:

> That the Board of Regents instruct the Council on Research to consider the establishment of minimal grants to members of the College for support of research in diseases of the chest.

Dr. Andrews stated that it is thought this procedure will significantly increase the amount of research sponsored by the American College of Chest Physicians, Council on Research, and will materially contribute to future fund-raising efforts. It is recognized that minimal research grants will be given the same attention by the Council on Research as though the grant were much greater.

It was further stated by Dr. Andrews that funds are required for the development of the Inhalational Therapy Association (for technicians) into a national organization. It is thought that the medical gas and equipment companies may be willing to give financial support to this and to the development of standards and certification through their trade organization, the Compressed Gas Producers

Association, Medical Committee. Approval is requested for the Council on Research to receive such funds and to disburse them under the direction of the Committee on Physiologic Treatment for the stated purposes.

Upon motion duly seconded the report of Dr. Andrews was accepted and the recommendations unanimously adopted.

Dr. Stygall presented a brief report on membership, stating that in the United States and possessions there were 2,647 members, in countries outside of the United States 1,659 members, and that 362 applications for membership had been filed since March, 1953, bringing the total membership of the College to 4,668. Dr. Kinsey, Regent of the College for Canada, remarked that the College membership in Canada had increased rapidly in the past few years and now had reached well over 200 members.

Mr. Kornfeld, Executive Director, discussed the proposed new application forms which had been prepared by the Committee on Credentials. He pointed out that the purpose of the new forms was to attempt to give the Regents and Governors more information concerning applicants for membership. The Board members were requested to study the new form and to send their comments and suggestions to the Executive Offices of the College in Chicago.

Dr. Myers reported briefly for the Editorial Board of the College journal, *Diseases of the Chest*. He pointed out that during the year 1953, a total of 145 papers have been published and that the present circulation of the journal is 6,500 copies monthly which reach 76 countries and territories throughout the world.

Dr. Banyai, chairman of the Council on International Affairs, announced that the Third International Congress on Diseases of the Chest would be held in Barcelona, Spain, October 4 through 8, 1954, and that literature pertaining to the Congress was being mailed to all members of the College. He stated that work had begun on the arrangements for the scientific program to be presented. It was pointed out that members who wish to present papers at the Congress in Barcelona should send their titles, as well as abstracts of their papers, to Dr. Andrew L. Banyai, care of the Executive Offices of the College in Chicago, as soon as possible. Complete manuscripts must be available at least six months in advance of the meeting. Mr. Kornfeld reported on his trip through Europe during the past summer and told of the splendid hospitality extended by College members in the countries he visited. He also reported on the organization of new College Chapters in many of the European countries, as well as the influx of new members in those countries.

The following resolutions were read:

WHEREAS There has been some criticism concerning the length of the program presented at the annual banquet of the College, and

WHEREAS This event should be planned in keeping with the social atmosphere which is desirable after a three day business and scientific session,

THEREFORE BE IT RESOLVED That the incoming President's address be given at the annual convocation of the College and that the time for his presentation be limited, not to exceed 15 minutes, and that the only authorized functions for the banquet be the following:

1) Introduction of officials and guests at the speakers table.
2) Presentation of the College Medal.
3) Presentation of the College Essay Award.

BE IT FURTHER RESOLVED That the introduction of officials and guests at the speakers table be expedited without extraneous remarks; that the presentation of the College Medal be limited to 10 minutes; and that the presentation of the College Essay Award be limited to 5 minutes. The balance of the evening should be given over to good fellowship and entertainment.

Upon motion duly seconded, the above resolution was adopted.

WHEREAS The American Association for the Advancement of Science has extended an invitation to the American College of Chest Physicians to officially affiliate with the aforementioned organization, and

WHEREAS Other recognized medical societies such as the American Medical Association, American Academy of Ophthalmology and Otolaryngology and American Public Health Association are affiliated societies of the American Association for the Advancement of Science, and

WHEREAS Affiliation with the American Association for the Advancement of Science carries no financial obligations, and

WHEREAS The objectives of the American Association for the Advancement of Science in no way contradict the objectives of the College,

THEREFORE BE IT RESOLVED That the American College of Chest Physicians formally affiliate with the American Association for the Advancement of Science.

Upon motion duly seconded, the above resolution was adopted.

Dr. Greer extended the appreciation of the Board of Regents to Drs. Goldman, Steiner, Kerr and Dunner and to the Missouri Chapter of the College for the splendid meeting presented in St. Louis. It was moved and seconded that the doctors named above be extended the appreciation of the Board in writing and that an expression of gratitude be sent to the Director of Barnes Hospital for his cooperation in arranging a tour and program at the hospital for the members of the Board of Regents. It was also recommended that the management at the Sheraton Hotel, St. Louis, receive an expression of appreciation from the Board.

Upon a motion introduced by Dr. Banyai, seconded by Dr. Myers, and unanimously adopted by the Board of Regents, Dr. Placak, who would be visiting El Paso, Texas on December 5, was authorized to extend to Dr. Hendricks the cordial greetings and best wishes of all of his friends in the College for a speedy recovery from his illness. The Board of Regents expressed regret upon Dr. Hendricks inability to be with them, as they have always enjoyed his companionship and have appreciated his wise counsel in guiding the progress of the College. It is extremely sad to report that only a week later, on December 9, Dr. Hendricks passed away in his sleep. Dr. Hendricks was a Founder of the American College of Chest Physicians and held membership Number 1. Words cannot adequately express the sorrow and deep sense of loss which is felt by the members of the Board of Regents, as well as many members of the College who knew Dr. Hendricks. He was a familiar figure at every annual and semi-annual meeting, as well as many regional meetings, which he attended since the inception of the College and up to the time of his illness. The members of the Board of Regents extend their deepest sympathy to Mrs. Hendricks and the immediate members of the family in their great loss.

Meeting adjourned.

DIAGNOSTIC CONFERENCE TO BE HELD AT ANNUAL MEETING

Dr. L. Henry Garland will conduct the Diagnostic Conference to be held at the 20th Annual Meeting of the American College of Chest Physicians in San Francisco, June 17 through 20, 1954. For all cases presented it is desired that complete diagnostic evidence be available, including history, x-ray films, bronchoscopy and bronchography when indicated, bacteriological and pathological findings. Physicians who wish to participate in this Conference are invited to submit their cases to Dr. Garland, 450 Sutter Street, San Francisco, California.

College Meetings in Germany

During his recent tour of Europe, Mr. Murray Kornfeld, Executive Director of the College, presented the following motion pictures, prepared by Fellows of the College, in 12 countries which he visited:

"Extra Periostal Lucite Ball Plombage for Pulmonary Tuberculosis,"
Overholt Thoracic Clinic, Francis M. Woods, Brooklin, Massachusetts.
"Endobronchial Tuberculosis,"
Paul H. Holinger, Chicago, Illinois.
"Surgical Treatment of Aortic Stenosis,"
Charles P. Bailey and Houck E. Bolton, Philadelphia, Pennsylvania.

Meeting in Wiesbaden, Germany, July 10

In Wiesbaden, Germany, these films were presented to members of the Verein der Arzt Wiesbadens at the New State Museum Building on July 10. The meeting was arranged by Professor Wurm, President of the Verein der Arzte Wiesbadens and Professor Paul Neumann, a Fellow of the College.

Professor Hugo Wilhelm Knipping, Director of the Universitat Klinik, Cologne, Germany and Mr. Murray Kornfeld, photographed during a College meeting at the Klinik.

Meeting in Hamburg, Germany, July 21.
Front row, far right, Professor Joachim Hein, Regent of the College for Germany.

Professor Hugo W. Knipping, Governor of the College for Germany, arranged a meeting on July 13 in Cologne at the Medical School of the University of Cologne at which approximately 85 physicians were in attendance.

On July 21, Professor Joachim Hein, Regent of the College for Germany, arranged a meeting sponsored by the Medical Society of Hamburg held at the University Hospital where more than 100 physicians viewed the films.

The films were also shown at meetings arranged by members of the College in Madrid and Barcelona, Spain; Leysin, Switzerland; Nurnberg, Germany; Amsterdam and Groningen, Holland; Malmo and Stockholm, Sweden; Oslo, Norway; Helsinki, Finland; Newcastle-upon-Tyne, England; Aberdeen, Scotland; and Liege, Belgium.

College Chapter News

PHILIPPINE CHAPTER

The annual meeting and installation of officers of the Philippine Chapter took place on August 20 at the Quezon Institute, Quezon City. The Honorable Juan Salcedo, Jr., Secretary of Health, inducted the new officers who are:

Sixto A. Francisco, Manila, President
Cirilo Santos, Quezon City, President-Elect
Laureano D. Bautista, Quezon City, Vice-President
Fidel R. Nepomuceno, Manila, Secretary-Treasurer.

PRESIDENT'S INAUGURAL ADDRESS

Conditions in the Philippines with regard to the tuberculosis problem are not unique. The last Pan Pacific Tuberculosis Conference showed us how similar are the difficulties found among most countries in the Pacific Area and Asia. Our Division of Tuberculosis of the Department of Health is engaged in a fundamental balanced control program as follows:

1) Prevention of tuberculosis by: (a) Health education, which is being conducted not so much by expensive printing of sparsely circulated written material, but by word of mouth, by loudspeakers, at barrio level; and (b) BCG vaccination. These are being undertaken extensively by field teams aided by UNICEF and WHO, and in a smaller scale in government chest centers and private institutions under government supervision.

2) Case-finding aided by x-ray films is done in existing chest centers and by mobile x-ray units, of which the Division of Tuberculosis has three.

3) Each government chest center is equipped with laboratory facilities to demonstrate tubercle bacilli, and to perform other routine examinations.

4) Hospital beds have not been given emphasis by the government program because of the great expense that would be needed for the benefit of relatively few. For public health purposes, it was felt that a limited budget is wiser spent in preventive measures and tuberculosis dispensaries. However, there are hospital beds set up specially for such cases receiving surgical treatment and collapse therapy. Under the MSA/PHILCUSA program the creation of tuberculosis wards in general hospitals in conjunction with chest centers rather than building expensive separate sanatoria, will be encouraged.

Despite the existence of these basic services, however, I have to candidly admit that the program of tuberculosis control, viewed as a whole, leaves much to be desired. Not alone because insufficient funds are allotted for the tuberculosis fight, but because these funds are divided among programs also divided, and among agencies, again, unfortunately divided.

In the Philippines, we had in 1947 a mortality rate of 164.70 per 100,000 and in 1952 we had on record 144.28 per 100,000 as our annual death rate from pulmonary tuberculosis.

EIGHTH ANNUAL POSTGRADUATE COURSE ON DISEASES OF THE CHEST

Registrants and lecturers who participated in the postgraduate course given in Chicago, September 28 - October 2, 1953.

With no other particular disease have the health authorities and the medical profession been squarely face to face with utmost difficulty than tuberculosis. The delegates of 30 countries within the Pacific area, assembled in Manila on April 18, 1953, recognized that tuberculosis is still a major medical problem in most of them and that there is a need for a periodic evaluation of the effectiveness of different methods of tuberculosis control. The WHO admits that no standard plan of tuberculosis control can be formulated which will fit every country even among the underdeveloped areas but it recommends international pooling of information so that such control techniques found effective in one country may be of great interest to neighboring countries and to the rest of the world.

The Philippines has, for the past 40 years, made a valiant effort to solve its tuberculosis problem, but there are meager indications that the disease is losing ground so that the goal for its control appears still remote. Let us honestly ask ourselves these questions: Do the effects of the 40-year-old campaign for the control of tuberculosis run counter to the efforts exerted and to the tremendous amount of money already spent? Or is the implementation of our admittedly fine antituberculosis plan practical, economical, systematic; and does it get the adequate financial support, the necessary cooperation, and mutual understanding among all the official and non-government health entities or agencies that are engaged in this campaign? The correct answers to these questions will place us on a solid footing enabling us to put our National Tuberculosis Campaign on more effective strategic ground. The results, no doubt, shall be more satisfying and lasting.

My friends, the Philippine Chapter of the American College of Chest Physicians has a decidedly important role to play in the solution of the problem outlined above. You have elected me to guide the affairs of this organization for this coming year, and I tremble somewhat at the great responsibility imposed upon me. My unequivocal policy shall be that of working for sincere, honest, and harmonious relations among all tuberculosis workers in the Philippines. Personal differences of opinion will always exist (otherwise, this already dull world of ours would be duller still).

I alone cannot succeed. My hands are powerless without yours to lend them strength. My voice alone is feeble without yours to lend it volume, so that in the aggregate expression of our mutual desires the first round in the successful fight against tuberculosis will have been won, which may eventually lead us to final victory.

Sixto A. Francisco.

EDITOR'S NOTE

Physicians working in diseases of the chest throughout the world were delighted with the return of the "Bulletin of the Quezon Institute" in June, 1953.

This Bulletin, which had been such a welcome visitor to the desks of such large numbers of physicians for so many years, was compelled to suspend publication in 1941 due to World War II.

The June, 1953, issue contains a fine array of articles on such subjects as bronchiectasis in Filipino children, treatment of pulmonary tuberculosis, results of three to eight month treatment with isoniazid in tuberculosis, and congenital diaphragmatic herna.

Dr. Miguel D. Canizares, Director of the Quezon Institute and Regent of the College for the Philippines, and his excellent staff have long made splendid contributions to the world's knowledge of diseases of the chest. This Bulletin has been greatly missed for the past 12 years. Dr. C. P. Jacinto, a Fellow of the College, is editor. Dr. Antonio G. Sison, chairman of the editorial board, and the members of the board plan to issue the Bulletin twice a year. Workers in diseases of the chest everywhere will do well to read every number.

J. Arthur Myers.

SIXTH ANNUAL POSTGRADUATE COURSE ON DISEASES OF THE CHEST

Registrants and lecturers who participated in the postgraduate course given in New York City, November 2-6, 1953.

NEW YORK CHAPTER

The New York Chapter will meet for its annual clinical session on February 18 at the Hotel New Yorker, New York City. Dr. Leonard C. Evander, Lockport, is the President. The following program, arranged under the direction of Dr. Ida Levine, Brooklyn, wil be presented:

Morning Session — 9:00 a. m.:

"The Problem of Mediastinal Tuberculosis,"
Harold A. Lyons, Brooklyn.
Discussor: I. D. Bobrowitz, Otisville.

"Industrial Diseases of the Chest,"
David Ulmar, New York City.
Discussor: Edgar Mayer, New York City.

"Progressive Spastic Bronchitis, Its Significance and Treatment,"
Maurice S. Segal, Boston, Massachusetts.
Discussor: Benjamin Burbank, Brooklyn.

"Clinical-Pathologic Conference: Diffuse Pulmonary Infiltrations,"
Charles E. Hamilton, Brooklyn.
Discussors: Eli H. Rubin, Bronx; Louis E. Siltzbach, New York City; and David M. Spain, Brooklyn.

12:00 noon — Luncheon Meeting:

"Clinical Value of Pulmonary and Cardio-circulatory Function Studies,"
Herbert C. Maier, New York City.

Afternoon Session — 2:00 p. m.:

"Non-inflammatory Rib Tumors,"
Lew A. Hochberg, Brooklyn.
Discussor: Bradley L. Coley, New York City.

"Pulmonary Complications of Cardiac Disease,"
Simon Dack, New York City.
Discussor: John B. Schwedel, New York City.

"Experimental Creation and Closure of Interatrial Septal Defects,"
Philip Crastnopol, Jamaica.
Discussor: Charles B. Ripstein, Brooklyn.

"Clinical-pathologic Conference: Localized Pulmonary Disease,"
Arthur Q. Penta, Schenectady.
Discussors: Coleman B. Rabin, New York City; Allan E. Bloomberg, Staten Island; and Oscar Auerbach, East Orange, New Jersey.

INDIANA CHAPTER

The Indiana Chapter, meeting in conjunction with the state medical association in French Lick, October 20, elected the following officers for 1953-54:

Edward W. Custer, South Bend, President
J. V. Sherwood, Fort Wayne, Vice-President
H. B. Pirkle, Rockville, Secretary-Treasurer (re-elected).

Dr. Otto L. Bettag, Chicago, Illinois, spoke on "Complications of Pneumoperitoneum," followed by an x-ray conference.

PACIFIC NORTHWEST CHAPTER

The annual meeting of the Pacific Northwest Chapter was held at the University of Washington Medical School, Seattle, November 14-15. The following officers were elected:

John E. Tuhy, Portland, Oregon, President
Herbert S. Stalker, Tranquille, British Columbia, Vice-President
Lawrence Lowell, Portland, Oregon, Secretary-Treasurer.

ARGENTINE CHAPTER

On November 14, the Argentine Chapter held its annual meeting in Buenos Aires and elected the following officers:

Oscar Cames, Rosario, President
Juan Rocca, Cordoba, Vice-President
Alfredo Cesanelli, Rosario, Secretary-Treasurer.

MEDICAL SERVICE BUREAU

POSITIONS AVAILABLE

Approved residencies for new 440 bed tuberculosis division of large general hospital; complete medical, surgical, and pathological facilities; excellent training program; medical school affiliation; east coast city; available now and also July 1. Please address all inquiries to Box 276A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Full time physicians wanted for new 440 bed tuberculosis division of large general hospital in east; available immediately; associated with two medical schools for teaching purposes; opportunity for research; complete surgical and pathological facilities; salary governed by background of training and experience. Please address all inquiries to Box 277A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Full-time resident wanted for cardio-respiratory laboratory on west coast. Work involves study of all types chest diseases, pre-operative evaluation for chest surgery and other surgical conditions, disability evaluation and the study of medical diagnostic problems. Salary $3,600 a year plus maintenance. Please address all inquiries to Box 278A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Applications will be received for positions of Resident and Assistant Resident at Grace Dart Hospital, 6085 Sherbrooke Street, East, Montreal 5, Quebec. Duties to commence July 1, 1954. Salary $3,000 and $2,400 respectively with full maintenance. All aspects of medical and surgical treatment of tuberculosis. 145 bed hospital approved by American College of Surgeons. Apply to Medical Director.

Senior staff physician wanted for 220 bed tuberculosis hospital using modern therapy including chest surgery. Annual stipend $6,552 to $7,800 with nominal deductions for apartment and maintenance. Based on qualifications, appointee may be started in second or third step. Apply Medical Superintendent, Pine Camp Hospital, Richmond, Virginia.

The Veterans Administration Hospital, Kerrville, Texas, a 449 bed tuberculosis hospital, has vacancies for an assistant surgeon, a chest physician (general practitioners eligible) and a pathologist. Starting salaries up to $10,800; positions based on the applicant's education and experience. Approved by American College of Surgeons and American Hospital Association, as well as possible residency training. Affiliated with diplomate consultants in the various specialties who make regular visits. In the hills of Southwest Texas, only 56 miles from San Antonio. Contact the Manager, Veterans Administration Hospital, Kerrville, Texas.

FOR SALE: Three lung equalizing chambers in perfect condition. For details, please address Box 275A, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

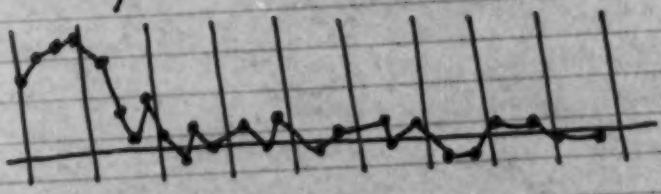
BLOOD PRESSURE STUDY IN PERSONS 65 AND OVER

There are increasing millions of people 65 and over, and it is important to learn: (1) What is the normal range of blood pressure for each age group over 65 for both sexes? (2) Does their blood pressure continue to increase with age? (3) Is the blood pressure related to weight, surface area, and height? (4) What is the relationship of hypertension to coronary sclerosis and occlusion, cardiac enlargement, valvular disease, calcification of the aorta? A similar investigation was already carried out in 74,000 gainfully employed people under 65 years of age.

Drs. Arthur M. Master and Harry L. Jaffe of New York have now undertaken a Blood Pressure Study in Persons 65 Years of Age and Over, under the sponsorship of the New York Heart Association. Industry and old age homes proved to be inadequate sources of the data required. Therefore, questionnaires were recently sent to 14,000 practicing physicians throughout the country. The cards are simple, clear, and can be completed by the insertion of the few figures requested, and by check marks in "yes" and "no" columns.

The response thus far indicates that many physicians are not aware that their cooperation is essential for the success of this project. Physicians who have received questionnaires are urged to complete them promptly. Those who wish to participate in the study may do so by sending a postcard to BLOOD PRESSURE, 11 EAST 100th STREET, NEW YORK 29, NEW YORK.

Dx/ Pneumonia



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Even cases that resisted previous treatment with other agents frequently show a gratifying response to Terramycin therapy. "A case of staphylococcal pneumonia, complicated by tension pneumothorax which had shown no response to [another antibiotic], made a rapid, complete recovery on a dosage of 15 mg. per lb."[3]

1. Knight, V.: New York State J. Med. 50:2173 (Sept. 15) 1950.
2. Fetterfield, T. G., and Starkweather, G. A.: J. Philadelphia Gen. Hosp. 2:6 (Jan.) 1951.
3. Swift, P. N.: Proc. Roy. Soc. Med. 44:1066 (Dec.) 1951.

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